SEPTEMBER, 1949

The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

CONVENTION NUMBER

Program, 14th Annual Convention and Course in Gastrointestinal Surgery

Pages I-XX

●

Fourteenth Annual Convention

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VOLUME 16 **NUMBER 9**

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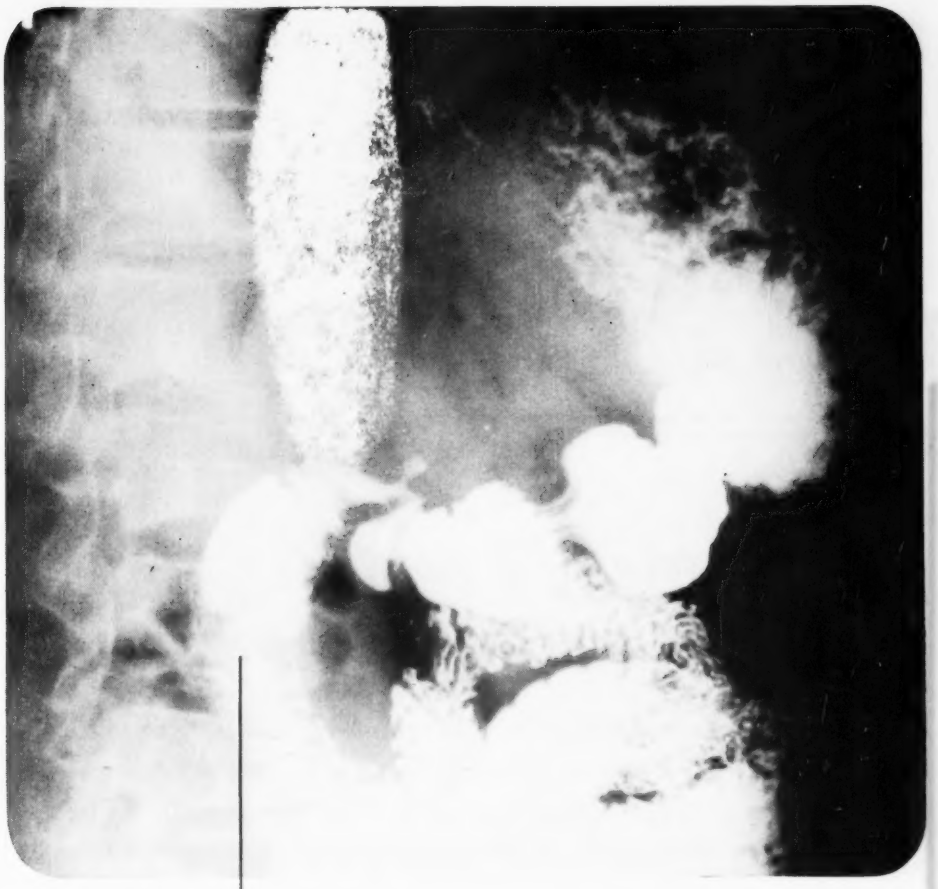

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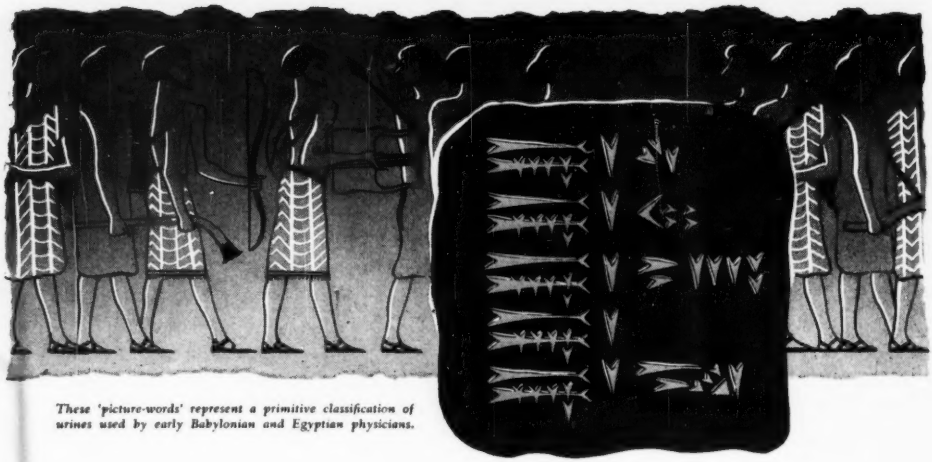

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The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

VOLUME 16 SEPTEMBER, 1949 NUMBER 9

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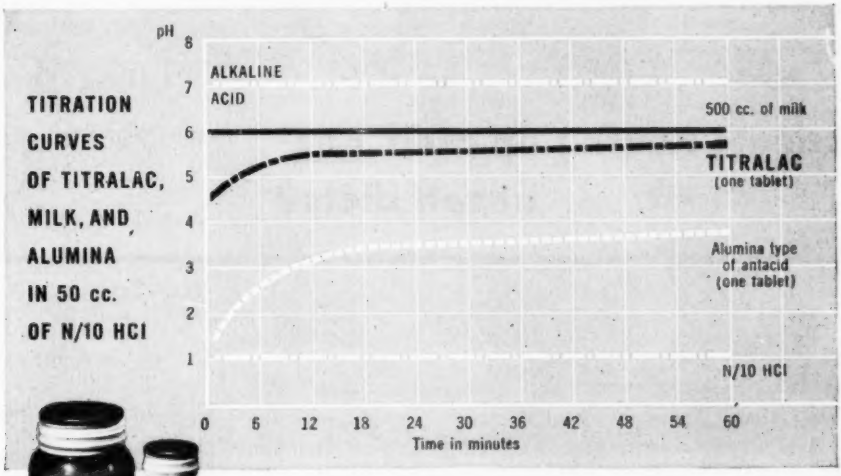

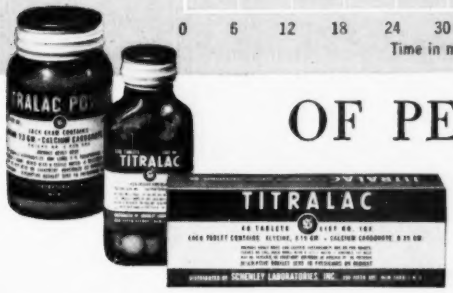

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1. Rossett, N. E., and Flexner, J.: Ann. Int. Med. 18: 193 (1944). 2. Freezer, C. R. E.; Gibson, C. S., and Matthews, E.: Guy's Hosp. Reports 78: 191 (1928). 3. Aaron, A. H.; Lipp, W. F., and Milch, E.: J. A. M. A. 139: 514 (Feb. 19) 1949. 4. Kirsner, J. B., and Palmer, W. L.: Illinois M. J. 94: 357 (Dec.) 1948. 5. Kimball, S.: in Practice of Medicine (Tice). Hagerstown, Md., W. F. Prior Company, Inc., 1948; p. 210. 6. Special Article: M. Times 76: 10 (Jan.) 1948.

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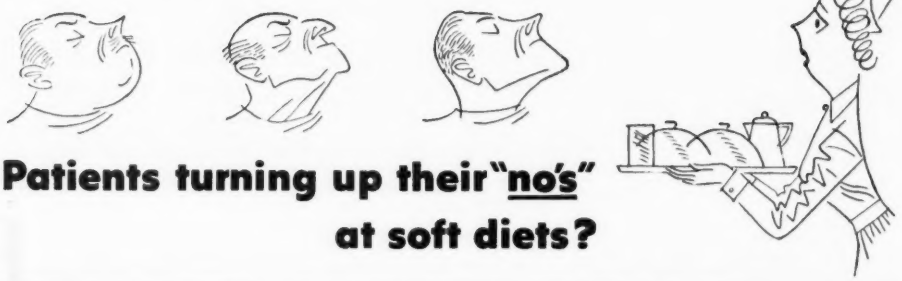

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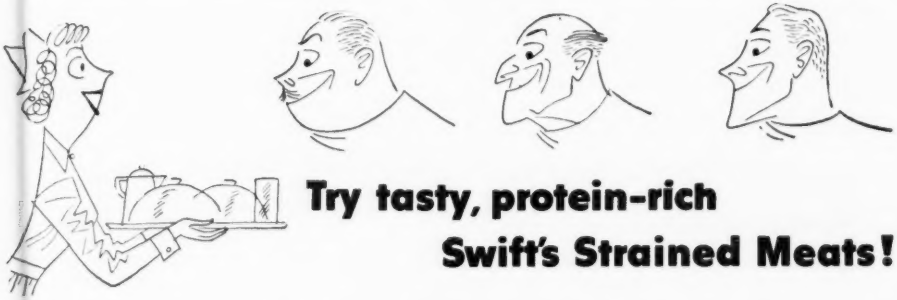

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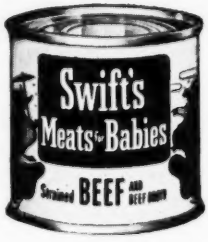

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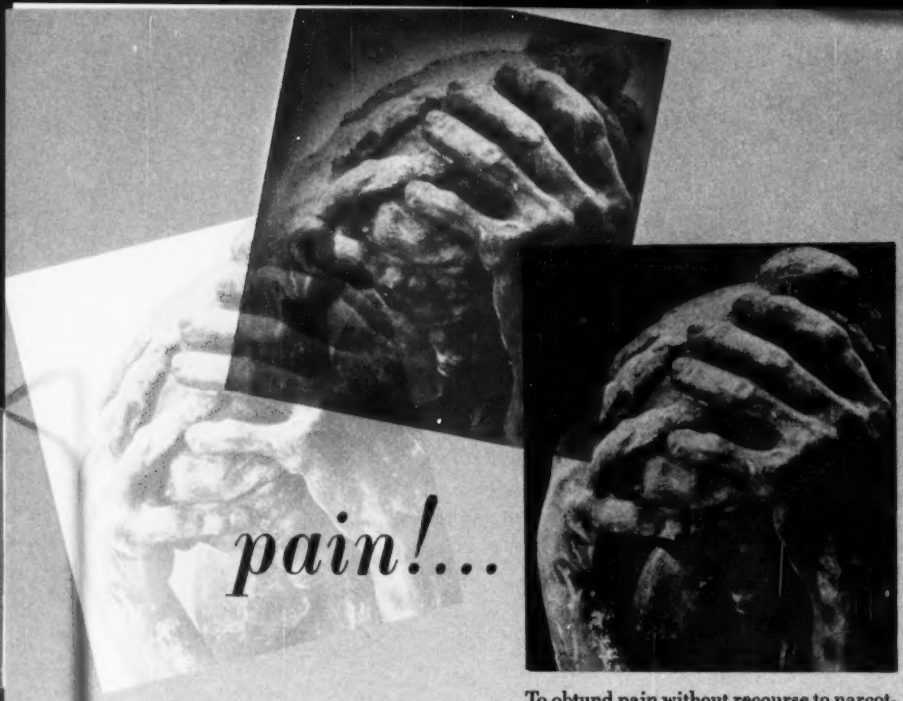

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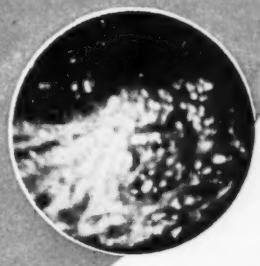

Gastrophotograph of mucosa coated by other substance.

1. Weiss, S., Espinal, R.B. & Weiss, J.: Therapeutic Application of Anion Exchange Resins in the Treatment of Peptic Ulcer, Review of Gastroenterology, 16:501-509, June, 1949.

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NEW PHYSIOLOGICAL AND CLINICAL STUDIES ON THE SECRETION OF MUCIN IN THE HUMAN STOMACH*†

GEORGE B. JERZY GLASS, M.D.**
New York, N. Y.

The available data on gastric mucin secretion in the human stomach are very scarce and confusing. Bockus[1] in his recent Max Einhorn lecture has stressed the urgent need for more information on the gastric mucin question to help to solve several gastroenterological problems.

During the last few years Dr. Boyd and I studied the mucin problem from several angles at the Department of Medicine of the New York Medical College and some new information was obtained on this subject. This concerns quantitative methods for determination of gastric mucin and its various fractions, differentiation and classification of various mucous components of the gastric content, their site of origin in the gastric mucosa, and their physiological and clinical significance[2-8]. Some of these data will be presented in this paper.

Figure 1 shows an outline of the various mucous components of the stomach, their classification and site of origin as we conceive it on the basis of our findings.

The total of the viscous substances secreted by the gastric mucosa and not derived from salivary or biliary contamination is here called "total gastric mucin". This is an inclusive name for two grossly different forms in which gastric mucin occurs: 1) visible gastric mucus, 2) dissolved gastric mucin.

Visible gastric mucus is found in the stomach as a) native slightly alkaline mucus which is jelly-like, tenacious and adherent to the gastric wall; b) if precipitated by hydrochloric acid of the stomach it forms lumps and shreds. These two forms together constitute the gastric visible mucus which is so familiar to all gastroenterologists.

The main carrier of the viscosity of the visible mucus is the substance which is herein called "mucoid of the visible mucus" or "surface epithelium mucoid". This contains a protein and a mucopolysaccharide moieties, and after imbibition with water and absorption of salts, enzymes and cells, forms the visible gastric

*Read before the New York Chapter of the National Gastroenterological Association, New York, N. Y., 14 March 1949.

†From the Department of Medicine, New York Medical College, Flower and Fifth Avenue Hospitals, New York City.

**Assistant Clinical Professor of Medicine, New York Medical College, Flower and Fifth Avenue Hospitals, New York City.

mucus. Concerning the site of origin of this substance most investigators agree that it is the secretory product of the columnar cells of the surface epithelium of the gastric mucosa[9].

The quantitation of the visible surface epithelium mucus in the clinic is practically impossible because it adheres closely to gastric mucosa and cannot be aspirated in toto. In addition, it has so many external similarities to saliva that many mistakes have been made in its gross estimation. For these reasons basic studies on gastric mucin can be done only on that part which Babkin and his collaborators[9] called "dissolved gastric mucin".

One can define dissolved mucin as the total of gastric mucous substances which are dissolved in the gastric juice, which confer upon it the feature of viscosity, and which cannot be separated from the fluid neither by centrifugation nor by filtration but by precipitation with alcohol or acetone. Two years ago we developed a method for the determination of this substance[2, 3], which was based on the principle of precipitation of the dissolved mucin with acetone from the trichloracetic acid filtrate of the gastric juice. The precipitate was taken up in alkali and the total dissolved mucin content was determined colorimetrically on the basis of the Folin-Ciocalteu phenol reaction, using tyrosine solution as a standard.

In further studies we found that the dissolved mucin is not an entity, as was universally accepted, but a heterogenous mixture of at least two different components, which we isolated[4].

The first of these substances we called "dissolved gastric mucoprotein" since we found it to be almost certainly indentical with the compound long ago isolated from the stomach of dog and given this name by Webster and Komarov[10]. Mucoprotein in its native state is dissolved in acid gastric juice, but we found that after precipitation with acetone it loses this solubility in the neighborhood of its isoelectric point, which is about 3.5, and separates out from the acid-salt solution at this pH. This made possible its isolation from the other fraction and quantitative determination by the Folin-Ciocalteu phenol reaction. Mucoprotein separates out in the form of light opal colored flocculi which are hydrated to a great extent and which after desiccation show a definite crystalline structure having a polygonal base. Its content of nitrogen and tyrosine is fairly high and of reducing substances rather low. It gives a violet biuret reaction.

The second component of the dissolved gastric mucin, which we isolated, we named "dissolved gastric mucoproteose" because it possesses features of a proteose as well as those of a viscous mucopolysaccharide. It differs in many respects from mucoprotein: It separates out in the form of heavy clumping resin-like particles, and not in the form of light flocculi. After desiccation it does not form a crystalline substance, but an amorphous white powder. After precipitation with acetone it remains soluble at pH 3.5, therefore at the range of pH at which the mucoprotein separates out. It can reprecipitated from this solution to great extent with acetone. It is partly soluble in 60 per cent alcohol. It usually gives a purple-violet biuret reaction, and it has much lower nitrogen and tyrosine and much higher reducing substances content than the mucoprotein.

What is the site of origin of these two mucous components in the gastric mucosa?

Concerning the gastric mucoprotein we have much evidence that it is secreted by the gastric glands and more specifically by the chief mucous cells of the neck of the glands located in the fundus and corpus of the stomach. Mucoprotein definitely has nothing in common with the secretion of the surface epithelium mucus. We were not able to find even a trace of mucoprotein in the visible surface epithelium mucus, nor in the native state, nor after splitting the visible mucus by enzymatic digestion or alkaline hydrolysis[5].

Additional reasons for suggesting its origin from mucous cells of the gastric glands are physiological and pharmacological considerations which will be discussed later, and the close similarity of mucoprotein to pepsin from the chemical, physiological and pharmacological point of view. Despite this there is no

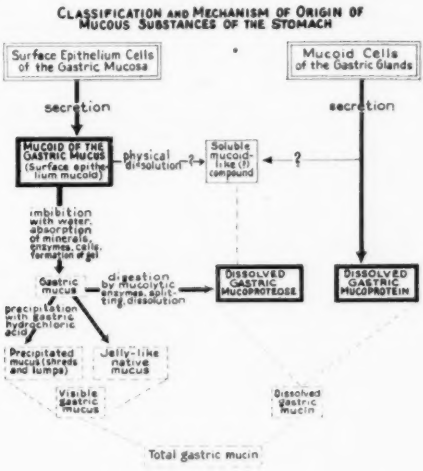

Fig. 1

doubt that mucoprotein and pepsin are different[11], but there may be some kind of complex linkage between them, as was suggested by Babkin and his associates[9].

The site of origin of the second component of the dissolved mucin, dissolved gastric mucoproteose, is quite different. We found[5] that the mucoproteose isolated from the fluid gastric juice was entirely identical with a substance which we obtained from the product of enzymatic digestion of the surface epithelium mucus, after having split the mucus by autodigestion in the incubator. On the basis of this unequivocal finding we advanced the evidence that the dissolved gastric mucoproteose was mainly the product of enzymatic digestion, dissolution and splitting of visible gastric surface epithelium mucus. Whereas the mucoprotein seems to be a definite entity, the mucoproteose shows fairly large range of variance in its composition. This suggests that it is a mixture of intermediate split products of visible gastric mucus in various transient stages of enzymatic digestion.

Some part of this mucoproteose complex may also derive directly from the surface epithelium, by physical dissolution of the mucus, or as a secretion of some other gastric glands, cardiac or pyloric, as shown in this graph. The main part of the mucoproteose is derived without any doubt, however, from splitting of the surface epithelium mucus.

What are the enzymes which split the visible gastric mucus into mucoproteose?

For 50 years it had been assumed that pepsin and hydrochloric acid were the main agents for dissolving the gastric visible mucus, and that in this way they might contribute to formation of peptic ulcer. We repeated these studies and found indeed that the pepsin in a low range of pH (about 1.5) contributes to the splitting of mucus; this resulted in formation of mucoproteose-like substances.

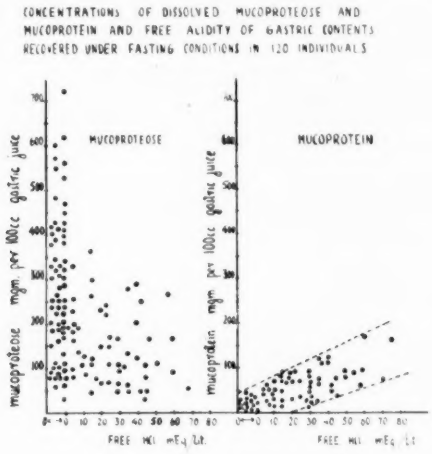

Fig. 2

In the last year Meyer, Prudden and associates[12-14] attributed a role to lysozyme as a main factor responsible for splitting of gastric and colonic mucus. In view of the great import of this statement for mucin studies we investigated the lysozyme problem from many aspects in the Laboratory of Dr. Stewart Wolf at Cornell University Medical College, with Mrs. Pugh's cooperation. We used the gastric mucus from the famous "Tom's" stomach and colonic mucus from patients with colonic fistula.

The details of these studies will be published separately[15]. I may only say, that we were not able to find any solvent action of lysozyme in vitro on the gastric mucus and its components, and we were unable as well to find any splitting action of lysozyme on the colonic mucus of man. We feel, therefore, certain that the lysozyme has nothing to do with the splitting of mucous substances of the human stomach and colon.

These are very discouraging findings in respect to the entire theory of Meyer and Prudden. We do not want to contest any of the facts which were discovered

in this connection, but we feel strongly that some revision of the interpretation of these findings must be made. Since we presented the evidence that the lysozyme is not the mucolytic enzyme of the gastric and colonic mucosa in man, it is probably necessary to revert back to the old status of lysozyme as described by Fleming and assume that its role in gastroenterology is mainly bacteriolytic and that the lysozyme deals only with the bacterial flora of the gastrointestinal tract.

It remains a fact, however, that the gastric mucus left alone in the incubator at the neutral range of pH undergoes liquefaction and digestion. Since at this pH the pepsin is inert and the lysozyme has turned out to be of no significance for this digestion, one must postulate that there is another mucolytic factor present, which acts at the neutral range of pH. This might be the trypsin or trypsin-like

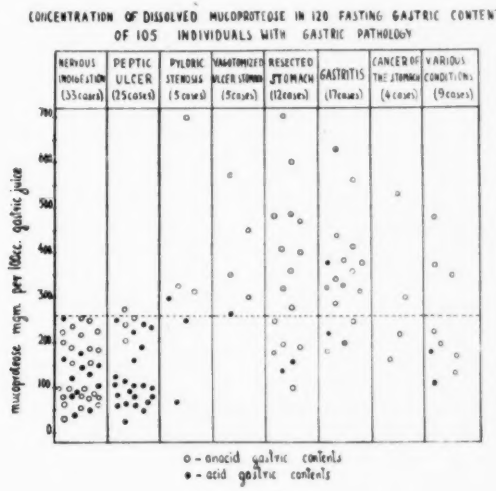

Fig. 3

enzyme, regurgitated from the duodenal content, or most probably another unknown gastric enzyme, which may be called "gastric mucolysine" although we do know nothing about its properties and characteristics.

In further investigations, at the Department of Medicine of the New York Medical College the dissolved mucin and its fractions were studied from pharmacological and clinical points of view[8, 16, 17]. Some of the results of these studies are shown in the following illustrations.

Figure 2 in the form of a scatter diagram shows the distribution of mucoproteose and mucoprotein concentrations in fasting gastric contents of 120 normal and pathological stomachs in relation to their free acidity. It is easy to note that the highest mucoproteose values were found in 20 fasting contents which did not show presence of acid, and that in contents in which the acidity and volume of secretion were high—the concentration of mucoproteose was rather smaller. There is inverse relation of mucoproteose values to acidity. This confirms once more

that the mucoproteose derives from surface epithelium mucus. Only this origin may explain why mucoproteose is found in highest concentrations in these contents which show absence of gastric acid secretion.

The range of mucoprotein concentration in the gastric content is much smaller than of mucoproteose, and it shows a fairly characteristic relation to gastric acidity. It is evident from Fig. 2 that in the anacid gastric content the mucoprotein is practically absent and that it is present in larger amounts in fasting contents with a night secretion of acid, like in ulcer stomachs. This behavior is just the opposite to that of mucoproteose, and suggests once more that the mucoprotein is the secretion of gastric glands.

Figure 3 shows the results of 120 determinations of gastric mucoproteose in 105 gastric contents recovered under fasting conditions. The horizontal line shows the upper limit of the normal range of mucoproteose concentration, which does not seem to exceed 260 mg. per 100 cc. gastric juice, in cases of acid as well as anacid gastric contents.

In peptic ulcer as well as in nervous indigestion, the gastric mucoproteose concentration is of the same range as that in normals. Since the output of the

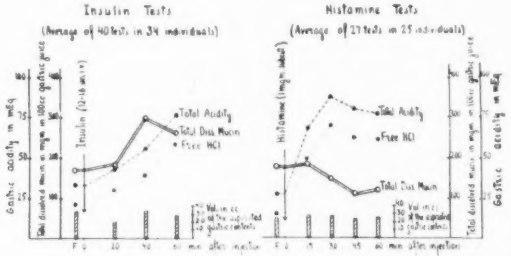

Fig. 4

gastric secretion in duodenal ulcer is certainly greater than in normals, we can assume that the mucoproteose output of an ulcer stomach is at least the same as in normals. Since the mucoproteose derives from mucus one can positively state that there is no mucus deficiency in the ulcer stomach as was erroneously suggested long ago.

Definite increase in mucoproteose concentration of fasting gastric content is found in 3 groups of cases: 1) in gastric retention, due to pyloric stenosis or to vagotomy; 2) in ulcer stomach after subtotal gastric resection; 3) in cases of gastritis (hypertrophic or atrophic). In gastric retention the increase in mucoproteose concentration depends probably on excessively prolonged interaction of substrate and splitting factor; in the gastrectomized stomach—on the decreased volume of diluting acid secretion and probably also on high mucus secretion, which is so often found after gastrectomy; in gastritis—on increased secretion of the substrate for mucolysis, i.e., on increased production of the surface epithelium mucus.

This latter finding seems to have vast clinical importance. If our further studies, which we are now doing with the gastroscopic cooperation of Drs. Barowsky and Schwartz will confirm these preliminary data, we might be able to make a "chemical diagnosis" of gastritis on the basis of a high mucoproteose level in gastric contents of these patients.

In further studies we tried to determine the effect of humoral and neural stimuli on the secretion and concentration of mucous components of the gastric juice. From a large series of tests, done with various stimuli[16, 17] the only figure which is shown here is the one that compares the effect of histamine, chief representative of humoral stimuli, and insulin, the outstanding representative of neural stimuli, upon the concentration of total dissolved mucin in the human stomach.

The histamine curve is drawn in Fig. 4 on the basis of 27 tests in 25 individuals with normal and pathologic stomachs. It shows that 1 mg. histamine causes

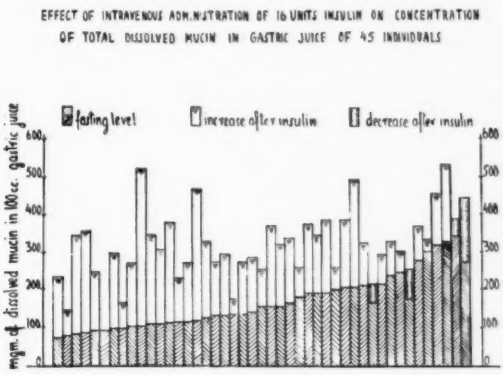

Fig. 5

a definite drop of the dissolved mucin concentration[8]. This coincides with the increased output of the acid gastric juice secretion after histamine.

On the other hand insulin, injected in a dosage of 12-16 units intravenously, in 40-60 minutes causes a definite increase in dissolved mucin level[16], which in average doubles the fasting value. This average insulin curve has been drawn on the basis of 40 tests in 34 various normal and pathological individuals. This rise coincides with the peak of the hypoglycemic symptoms, but precedes by about 20-40 minutes the peak of increase of acidity, which usually occurs only about 1½ hours after insulin injection.

The uniformity of the dissolved mucin response to insulin is shown in Fig. 5, in which each column represents one insulin test. The shaded areas show the average fasting levels, and the unshaded areas—the increase in dissolved mucin 40-60 minutes after intravenous injection of insulin. Only in 4 cases, showing high initial fasting levels, did we find a drop in dissolved mucin after insulin. In the remaining 41 cases, the results uniformly showed a definite increase in dis-

solved mucin concentration after insulin, which has proved to be statistically highly significant, the P value being below 0.001.

After having differentiated dissolved mucin in its two fractions we studied, in further series of investigations, the effect of humoral stimulation with histamine, and neural stimulation with insulin on the mucoprotein and mucoproteose fractions separately. The mucoprotein and mucoproteose curves in Fig. 6 represent the average from 6 tests, calculated with standard errors, in which histamine and insulin were injected one after the other at intervals of 45-60 minutes. It should be noted that 1 mg. histamine causes only a very slight increase in mucoprotein concentration which lasts for 15-30 minutes. This response is very similar to that of pepsin after histamine[9]. On the other hand, the mucoproteose fraction after histamine shows a definite drop in its concentration synchronous to the rise of gastric acidity.

AVERAGE CURVES OF MUCOPROTEIN, MUCOPROTEOSE AND FREE ACIDITY OF THE GASTRIC JUICE AFTER HISTAMINE – INSULIN INJECTION. (MEANS OF 6 TESTS CALCULATED WITH STANDARD ERROR)

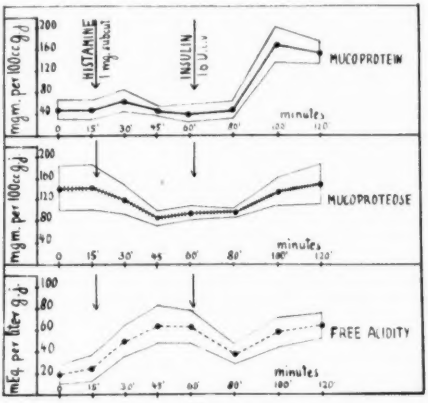

Fig. 6

The effect of insulin on mucoprotein is very manifest. There is a sharp rise in concentration of mucoprotein 40-60 minutes after i.v. injection of 16 units insulin. The average concentration of mucoprotein after insulin is more than tripled in comparison with fasting levels. The effect of insulin on the second mucin fraction—mucoproteose—is less significant and not constant, so that there is, on the average, only a slight rise above the fasting level.

The increase in mucoprotein after insulin is uniformly found in all normal stomachs as well as in these with peptic ulcer, in further series of our investigations. This shows that the rise in total dissolved mucin concentration which we found previously after insulin injection, was due mainly to the stimulation of mucoprotein secretion with insulin. This is evident from the Fig. 7 in which is indicated the composition of the dissolved mucin in regard to both its fractions,

depending on conditions under which the gastric juice was recovered. One can readily note that the dissolved mucin recovered from fasting anacid stomachs is composed mainly of mucoproteose (white columns), whereas mucins recovered from the acid gastric contents after vagotropic stimulation with insulin and mecholyl are composed mainly from mucoprotein (shaded columns). The fasting acid contents show an intermediate position in this respect[4].

Therefore the mucoprotein fraction of the dissolved gastric mucin seems to be strongly under the influence of nervous impulses. These are carried to the stomach by the intermediary of vagus nerves as the result of stimulation of vagal centres by insulin hypoglycemia[16] or peripheral parasympathetic stimulation with mecholyl[17].

Since the response of gastric secretion to vagal influence depends on the integrity of nervous pathways to the stomach, it might be anticipated that the secretion of mucoprotein after insulin would be abolished if the vagi nerves were cut, as it is the case in the vagotomized stomach.

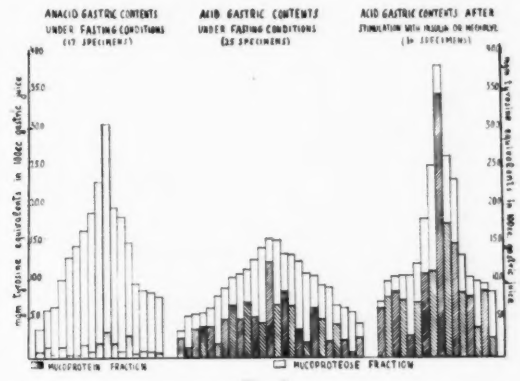

Fig. 7

Figure 8 shows that this is really the case. The average curves of mucoprotein and acid secretion are drawn in Fig. 8 on the basis of data obtained in 20 nonoperated peptic ulcers, 13 subtotally resected ulcer stomachs, and 6 vagotomized stomachs, operated for duodenal ulcer*.

This figure brings the evidence of a strong positive response of gastric mucoprotein to insulin in nonoperated duodenal and juxta-pyloric ulcers, which coincides with the insulin hypoglycemia. The mucoprotein values are here, on the average, more than tripled after insulin. It is also evident that the increase in the mucoprotein concentration in the gastrectomized stomach is entirely the same

*Most of these operations were done in the Department of Surgery of the New York Medical College, and I should like to express my thanks to Dr. James M. Winfield, the Head of the Department of Surgery for his help and interest in these studies. I should like also gratefully to acknowledge the cooperation of the members of the Surgical Staff of Flower and Fifth Avenue and Metropolitan Hospitals, and of Dr. Svigals from the Department of Medicine of the New York Medical College.

as for the nonoperated ulcer. This is obviously due to the fact that in the gastrectomized stomach the fundus area, which is responsible mostly for the mucoprotein secretion, is preserved.

On the other hand the completely flat curve of mucoprotein after insulin in the vagotomized stomach should be noted. The vagotomized stomach shows a depression of the secretion of gastric mucoprotein and does not respond to the insulin with the increase in mucoprotein, which is so typical for the normal stomach[8, 16]. This is similar to the behavior of the acidity curve after vagotomy, described by Hollander[18, 19].

It is clear therefore, that the mucoprotein test may supplement Hollander's acidity test, but may also add some new information which cannot be obtained

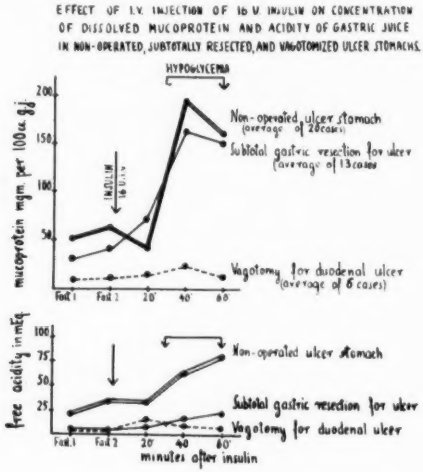

Fig. 8

by the testing of acidity only. This will be evident from the following consideration: The acidity response to insulin is abolished or on a low level both in vagotomized and resected stomachs, as is known to all surgeons, and as is also shown in our graph. If vagotomy is associated with subtotal gastric resection the negative response of the acidity to insulin may depend upon the vagotomy as well as upon the resection itself, as has been stressed recently[20, 21]. In such cases the response of mucoprotein to insulin, which is abolished by vagotomy but not by the resection, may give the clue to the interpretation of the negative Hollander's acidity test. The negative response of acidity and preserved response of mucoprotein to insulin would indicate that the negative Hollander's test under this circumstance was due mainly to gastrectomy, and not to vagotomy, and that some intact nerves still run to the stomach. On the other hand the negative response of both acidity and mucoprotein to insulin would indicate that the nervous pathways to the stomach were severed completely.

In view of these findings it is suggested[16] that testing of mucoprotein response to insulin be routinely added to Hollander's test for evaluation of completeness of vagotomy and integrity of nervous pathways to the stomach.

Figure 9 shows the difference between the vagotomized, resected, and nonoperated ulcer stomachs in regard to mucoprotein secretion after insulin. The data are not calculated on the basis of concentrations as in the previous figure, but on the basis of total output of mucoprotein in 20 minute samples. The curves again represent averages from many tests. Highest outputs of mucoprotein secretion after insulin in nonoperated stomach can be seen. Much lower output is found in the resected stomachs. This is due to the decreased rate of secretion of gastric juice after the operation, which causes the lowering of the output of mucoprotein in spite of its high concentration level. The lowest output, practically none, is found in the vagotomized stomachs. This confirms the previous inferences.

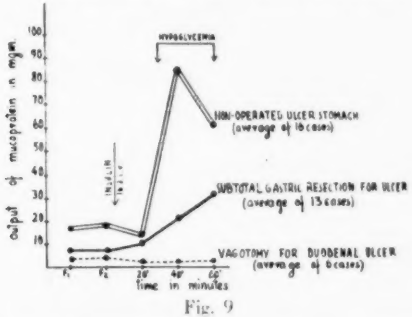

Fig. 9

On the basis of these findings we can assert that the mucoprotein is a component of the gastric secretion which is most exclusively dependent upon the vagal influences carried to the stomach from the brain. Since the effect of humoral influences on mucoprotein is almost nil, as shown by histamine tests, studies on mucoprotein secretion seem to be more adequate for the purpose of evaluation of vagal influences carried to the stomach, than studies of acidity, which depend on so many other factors and humoral stimuli. I personally believe that the testing of gastric mucoprotein after insulin stimulation may have the same significance for evaluation of vagal influences on the stomach, as the study of the gastric acidity after histamine has for estimation of the humoral phase of gastric secretion. For this reason the quantitation of mucoprotein concentration in the gastric juice is most adequate for the purpose of evaluation of emotional and other psychic influences on the human gastric function, together with other signs described by Wolf and Wolff[22].

The second main factor upon which the response of mucoprotein to stimulation depends is the status of the effector organ, i.e., of the mucous cells of the

gastric glands, which are responsible for mucoprotein secretion. The acidity response to humoral stimuli depends on the status of parietal cells of the gastric mucosa and of cells in the antral area in which the gastrin is elaborated. In the same way the response of gastric mucoprotein to insulin would depend on the status of mucous cells of gastric glands located in the fundus and corpus area of the stomach, provided that the vagi nerves are intact. We should expect that a diffuse lesion or atrophy of gastric glands would decrease or abolish the secretion of mucoprotein and its response to insulin.

In Figure 10 are curves of mucoprotein and acidity after insulin based on 20 peptic ulcer and nervous indigestion cases and 7 cases of atrophic gastritis and diffuse carcinomatous infiltration of the stomach, verified by gastroscopic or histologic examination of resected specimens.

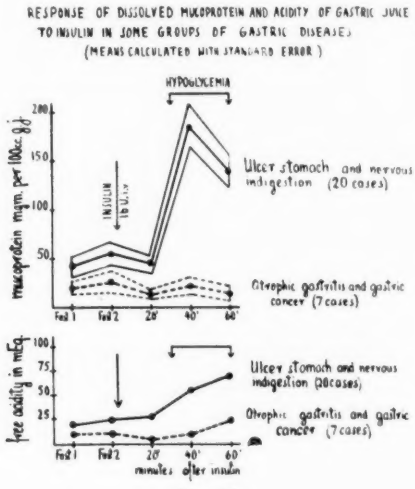

Fig. 10

In this figure, two definite and entirely different patterns of acidity and mucoprotein response to insulin can be noted. One, which we call "positive pattern" is characterised by positive response of mucoprotein and acidity to insulin. We find this positive pattern in normal cases, and also in cases of nervous indigestion and duodenal ulcer; in the latter the mucoprotein response is even higher than in normal cases. The second pattern which we call "negative pattern" is characterised by low or no response of acidity and mucoprotein to insulin. It seems to be typical for cases of diffuse atrophic gastritis[16], and for vagotomized stomach, as shown before. We found the same pattern in two cases of diffuse carcinomatous infiltration of the stomach. These data provide the evidence that the testing of mucoprotein response to insulin is important for the testing of the secretory activity of mucous cells of gastric glands.

Finally we can distinguish the third type of response, which we shall call "dissociated pattern" and which is characterised by negative or low response of acidity, and by a definite positive response of mucoprotein to insulin. This pattern is shown in Fig. 11, and the ascending curves of mucoprotein and the almost flat curves of acidity may be readily noted after injection of insulin. The dissociated pattern, as mentioned before, is found in cases of subtotally resected stomach. The explanation of this is mostly topographic. The antral area producing gastrin and containing part of parietal cells is resected, hence the low response of acidity to insulin. The fundus and upper corpus area producing mucoprotein is left, hence positive response of mucoprotein to insulin.

The same type of response can be expected in the nonoperated stomach, if the lesion has the same topographic location and invades the antral area leaving

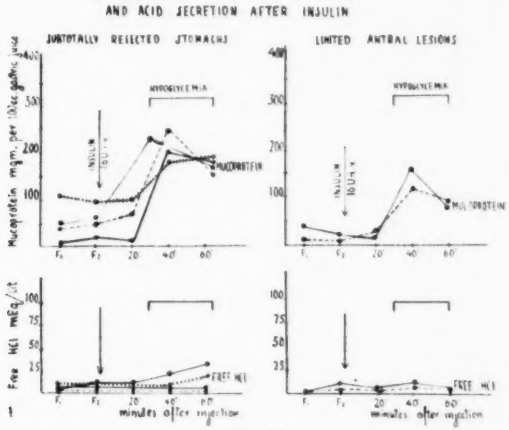

Fig. 11

the fundus intact. This dissociated pattern we observed in two nonoperated stomach cases, drawn in Fig. 11. Without knowing anything about these patients we assumed by analogy that there must have been a diffuse lesion of the prepyloric and antral area without invasion of the upper part of the stomach. On the operation in one of these cases atrophic lesions of the antral region were found associated with pyloric ulcer; the second case turned out to be cancer of the antrum. Fundus and corpus of the stomach were intact in both cases*.

The data presented in the last few figures propound the evidence that the mucoprotein test gives such a type of information which cannot be obtained by means of other available tests. In order to enable more extensive studies on this subject we have recently simplified the technic of mucoprotein determination and in place of colorimetric Folin-Ciocalteu reaction[7] have substituted a very simple volumetric determination of the precipitate of mucoprotein recovered from the

*Three other similar cases have been recently observed.

gastric juice[16]. The results of this simplification were good and made it possible to use the mucoprotein test as an office procedure.

The insulin injection itself, if carried out with small doses of insulin (12-16 units i.v.) did not give us, in about 100 tests which we have performed, any important side reactions, except the usual drowsiness, dizziness, perspiration, and feeling of hunger.

Before concluding this paper, the importance of differentiation of gastric mucin into its various components must be stressed. Only this differentiation made it possible to quantitate mucous substances more exactly and to trace their physiological and clinical significance. It is obvious that only future studies on a larger scale will allow us to determine the applications and limitations of the new mucin studies for physiological and clinical research.

It is, however, already evident that the quantitative determination of muco-proteose fraction is significant for evaluation of the secretion of surface epithelium of the stomach, and it may replace the quantitation of visible gastric mucus and help in the clinical diagnosis of gastritis. On the other hand the quantitation of the mucoprotein is important for evaluation of the vagal influences upon the stomach. In addition the testing of the mucoprotein response to insulin gives information on the integrity of gastric nervous pathways and the secretory activity of gastric glands located in the area of the fundus and body of the stomach.

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THE TECHNIC OF ABDOMINOTHORACIC TOTAL GASTRECTOMY USING THE GRAHAM METHOD OF ESOPHAGOJEJUNAL ANASTOMOSIS*

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The purpose of this paper is to describe the technic and report a case of total gastrectomy and partial esophagectomy using the Graham method of esophagojejunostomy through a combined abdominothoracic incision.

Allen (1938)[1], Lahey (1938)[2], Graham (1940)[3], Morton (1942)[4], Pack and McNeer (1943)[5], Orr (1947)[6], and Priestley and Kumpuris (1948)[7] have reported cases of abdominal total gastrectomies. Carter (1947)[8] performed a splenectomy through a combined thoracoabdominal incision and commented upon the excellent exposure. Henle (1909)[9], Wendel (1910)[10], Janeway and Green (1910)[11], and Ochsner and DeBakey (1941)[12], have reported on partial esophagectomies and gastrectomies done as two-stage abdominothoracic or thoracoabdominal procedures. Ohsawa (1933)[13], Brock (1942)[14], Loucks and Wu (1942)[15], Humphreys (1946)[16] and Garlock (1946)[17] have performed partial esophagectomies and gastrectomies as one-stage procedures through a combined abdominothoracic incision. Thorek (1948)[18] has mentioned the feasibility of doing a total gastrectomy through a combined thoracoabdominal incision, and Reynolds and Young (1948)[19] have recently reported on the performance of total gastrectomy and partial esophagectomy through a combined abdominothoracic incision utilizing the Roux Y technic of esophagojejunostomy.

Technic

Step 1. Skin Incision and Abdominal Exploration:—

The patient is placed on his right side and the operating table tilted 10° backward. The right knee is bent, the arms brought overhead and the pelvis strapped to the table. Make a left upper rectus muscle-splitting incision from the 7th costal cartilage to the umbilicus. Open up the peritoneal cavity and explore its contents. Palpate the gastric tumor and determine its size, mobility and metastasis to the regional nodes, liver and adjacent organs. A solitary metastatic liver nodule or nodal involvement along the greater or lesser curves of the stomach should not be considered contraindications for surgery.

Operability determined, extend the abdominal incision over the costal arch and then continue it along the 7th or 8th rib to the interscapular area. Resect the underlying rib subperiostally from its neck to the costochondral junction and then transect the costal arch. Incise the pleura and join the pleuroperitoneal

*From the Department of Surgery of Tufts College Medical School and the First Surgical Service of the Boston City Hospital.

cavities. The intercostal vascular bundle may or may not be ligated and intercostal nerve injected or resected.

Step 2. Incision of Diaphragm:—

Inject or crush the phrenic nerve at the cardiophrenic angle to paralyze the diaphragm. Incise the diaphragm radially, from the costal margin to the esophageal hiatus.

Step 3. Mobilization of Esophagus:—

Cut the inferior pulmonary ligament, retract the lung medially and locate Truesdale's esophageal triangle (heart, diaphragm and aorta). Incise the medi-

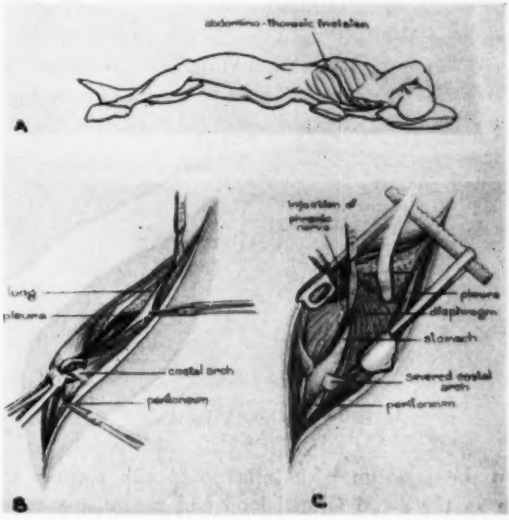

Fig. 1—A. Patient in left lateral position and skin incision extending from umbilicus onto chest along 7th rib.
B. Division of costal cartilage and formation of a common pleuroperitoneal cavity.
C. Radial incision of diaphragm from costal arch to esophagus and injection of phrenic nerve.

astinal pleura in this triangle a short distance along the lower ⅓ of the esophagus. Hook a finger beneath the esophagus and mobilize it by blunt and scissor dissection for at least 4″ or 2″ above the tumor.

Step 4. Splenectomy:—

Remove the spleen if its pedicle or belly is involved with tumor. If splenectomy is indicated, reflect the spleen medially to expose its posterior surface and the true pedicle. Divide the lienorenal ligament and ligate the splenic artery and vein individually. After the blood supply to the spleen has been shut off, return the spleen to its bed and divide the presplenic fold and gastrosplenic ligament. This maneuver exposes the ligated splenic vessels from in front and their division is easily accomplished.

Step 5. Mobilization and Transection of Stomach:—

The stomach is completely mobilized from its esophageal junction down to the pylorus, by division of the gastrohepatic and gastrocolic ligaments. The left gastric artery is divided near the celiac axis, and the right gastric and right gastroepiploic vessels are divided near the pylorus. The left gastroepiploic vessels may be severed if they have not already been cut during splenectomy. Transect the stomach in the region of the pylorus between Wangensteen clamps and wrap a rubber sheet or gauze around the cut end of the proximal portion of the stomach. Close the duodenal stump with two rows of inverting continuous g.i. 000 chromic catgut sutures and one layer of interrupted 0000 silk Halsted sutures.

Step 6. Esophagojejunal Anastomosis:—

Reflect the stomach cephalad and divide the vagi near the cardioesophageal junction. If the esophagus needs to be additionally mobilized, it is done at this time. About 24" from the ligament of Treitz pull up a loop of jejunum either ante- or retrocolically and lay it near the noncancerous portion of the esophagus. Anchor

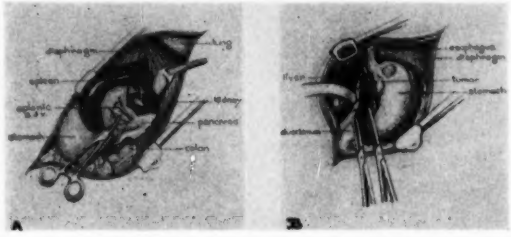

Fig. 2—A. Splenectomy. B. Mobilization of stomach by division of gastrohepatic and gastrocolic ligaments and transection of the duodenum.

the esophagus to the jejunum with interrupted silk sutures and then lay the esophagus on top of the distal jejunal loop and secure it there with several silk sutures. Several supporting sutures are also placed between the jejunum and the diaphragm. Again reflect the stomach superiorly and perform an open anastomosis between the esophagus and distal jejunal loop using an outer posterior and outer anterior row of interrupted 0000 silk, and an inner posterior and inner anterior row of interrupted 000 g.i. chromic catgut. During the performance of the anastomosis, the esophagus is transected above the tumor, the specimen discarded, and a Levine tube inserted into the distal jejunal loop. With the anastomosis completed, fold the proximal jejunal loop over onto the esophagus, and the distal jejunal loop, and anchor it to these structures with interrupted silk. To take tension off the anastomosis, the jejunal loop is sutured to the mediastinal and parietal pleura.

Step 7. Enteroenterostomy and Closure of Diaphragm:—

About 6" below the esophagojejunal anastomosis, perform an open entero-enterostomy between the proximal and distal jejunal limbs. Close the diaphragm loosely with mattress sutures around the intrathoracic jejunal loops.

Step 8. Closure of Thoracoabdominal Wound:—

Close the chest wall in layers around two catheters for under water drainage and close the abdominal wound without any drains.

Case Report

A. B., a 62 year old white female was admitted to the Boston City Hospital with symptoms of nausea, vomiting, substernal pain, weakness, melena, and weight loss of 4 months duration. Physical examination revealed marked anemia, arteriosclerotic heart disease, negative lungs and slight midepigastric abdominal tenderness. No masses were palpable in the abdomen or rectum. Vaginally, patient had a procidentia.

Laboratory data showed 2,300,000 red blood count; 20 per cent hemoglobin; 9,100 white blood count; differential blood count of 54 per cent polys, 7 per cent monocytes, 38 per cent lymphocytes and 1 per cent eosinophiles; negative urine; guaiac positive stools; FBS 95; NPN 31; total protein 6.5 per cent; Cl 91 me/1;

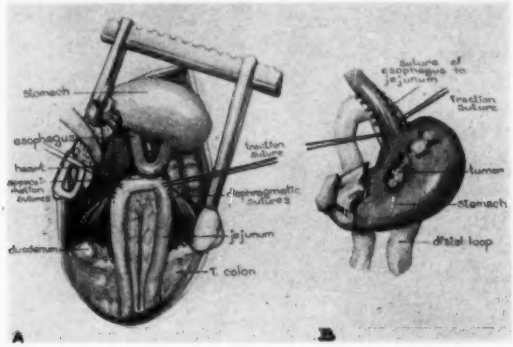

Fig. 3—A. Approximation of jejunal loop to esophagus and placement of supporting sutures to diaphragm.
B. Suture of esophagus to distal loop of jejunum.

CO_2 41 per cent; prothrombin time 85; 0 free HCl before histamine and 10 u. after histamine. Gastrointestinal series demonstrated an irregularity along the lesser curve of the stomach and a narrowing of the lower 3 cm. of the esophagus, consistent with carcinoma. Chest plate was negative for metastatic lesions.

The patient was treated preoperatively with vitamins, whole blood transfusions, intravenous salt, glucose, and amigen solutions and intragastric feedings with intermittent suction through a Levine tube. About 2½ weeks after admission, the patient was ready for operation with a red count of 3,100,000 and a hemoglobin of 12 g. Under cyclopropane, oxygen, ether endotracheal anesthesia, total gastrectomy, partial esophagectomy, splenectomy and omentectomy were performed through a combined thoracoabdominal incision. The operative procedure is essentially the same as is described and illustrated under technic. Pathological examination of the resected specimens revealed a large fungating nodular mass of the lesser curve and anteroposterior walls of the stomach which spread

to the lower few cm. of the esophagus, several lymph nodes along the lesser curve and periesophageal region were involved with carcinoma. Microscopically, the diagnosis was adenocarcinoma of the stomach with invasion of the submucosa of the esophagus and metastasis to the regional lymph nodes. Spleen and omentum negative.

Postoperatively, the patient was treated with nasal O_2, parenteral infusions of whole blood and amigen, vitamins, salt and sugar solutions, and constant intragastric suction. Penicillin and streptomycin were administered intramuscularly, and intrapleurally through one of the chest catheters. The left chest was tapped several times for fluid. An empyema of the left pleural cavity developed, the

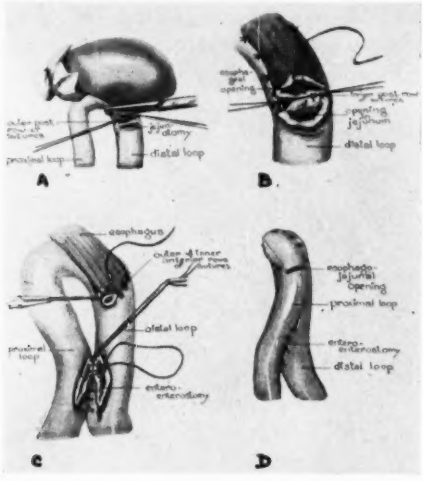

Fig. 4—A & B. Esophagojejunal anastomosis performed by using outer posterior and outer anterior rows of interrupted silk sutures and inner posterior and inner anterior rows of interrupted catgut.
C. Creation of enteroenterostomy between proximal and distal jejunal limbs.
D. Peritonealization of esophagus by overlapping of proximal jejunal limb onto distal jejunal limb.

exudate of which cultured B. coli. The empyema was treated with an intermittent saline drip apparatus containing penicillin and streptomycin. On the 5th postoperative day, the patient was placed on Mason's regime and a soft solid diet the 14th postoperative day. A check barium swallow revealed no intrathoracic fistula and an adequately functioning anastomosis. The patient was discharged 2 months after admission, still draining a small amount of purulent discharge.

The patient was followed in the outpatient clinic and 6 months after operation, x-rays revealed a slight narrowing of the esophagojejunal anastomosis probably due to recurrence. The patient's appetite remained poor and nutrition maintained mostly by liquids, oral protein, hydrolysates and vitamins.

At this point, some of the technical aspects of abdominothoracic total gastrectomy should be discussed. Rib resection versus intercostal thoracotomy is optional, and the same is true for injection or resection of the intercostal nerve exposed in the thoracotomy incision. To insure immobilization of the diaphragm, the phrenic nerve may be crushed or injected. The use of a cautery, carbolized knife and crushing clamps should be avoided on the segments of esophagus and jejunum to be anastomosed. In performing the anastomosis, a three-layered closure is better than a two-layered closure and interrupted sutures are preferable to continuous sutures. The graham technic is used because it peritonealizes the distal portion of the esophagus and reinforces the anastomosis with additional bowel wall. An enterostomy should be formed between the two jejunal loops because the proximal loop is obstructed when it is overlapped onto the esophagus, and distal loop of the jejunum. The loop of the jejunum should be supported from the mediastinal pleura, diaphragm, parietal pleura and connective tissue which overlies the vertebrae and ribs, to take the tension off the anastomosis. Catheter drainage of the pleural cavity is advocated over nondrainage because of the danger of leakage of intestinal contents into the pleural cavity.

Addendum

Since submitting this report for publication, two additional cases of carcinoma of the stomach were treated by the technic of abdominothoracic total gastrectomy using the Graham method of esophagojejunal anastomosis.

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BIOTOXIC INTESTINAL CONDITIONS OF THE ACID FERMENTATION TYPE*

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In Herter's[1] book the saccharo-butyric (acid fermentative) type of intestinal putrefaction was advanced. Schmidt and others had, by deduction, suggested the condition from tests of human feces. Much has been learned about the condition since Herter's description, (which occurred at an early date) and our knowledge of intestinal bacteriology has increased greatly.

Under the thirteen generic headings of intestinal organisms 171 subdivisions have been described. Of these, the strict anaerobes are in the minority as compared to the aerobes, and, while symbioses of both types of micro-organisms are important in the production of toxic states, the anaerobes are more so in the causation of this type of toxemia. They occur particularly in the colon and lower ileum and generally are the spore bearing organisms. The anaerobes, particularly important in the condition, are those belonging to the generic groups of bacteroides and clostridiums. The enterococci B, Gram positive diplococci and several of the pathogenic aerobes are usually associated with them. In many instances of the condition the coliform organisms are replaced, often being absent in Gram stained stool specimens. Still, very acid stools may be seen in which the coliform organisms are pronounced and gas production on medias abundant. It is interesting that the aerobes can multiply in the bowel in such large numbers in the absence of, or with very slight amounts of oxygen and conversely that anaerobes may be abundant with considerable oxygen in the colon. This irregularity to laboratory experience cannot be explained. In the definitely established condition, anaerobiosis may be quite complete. Heat, darkness and moisture encourages their growth, especially if enough carbohydrates and fats are present in the bowel contents.

Chronic excessive acid fermentative conditions are wide-spread among adults. In children they are relatively uncommon. The amount in which this condition is present varies within wide limits. The anaerobes are in the intestine of nearly all adults, and there are probably few persons who, from time to time, do not suffer slight temporary derangements of intestinal digestion associated with the temporary multiplication of these anaerobes which, as stated, usually are found associated. The condition does not seem to shorten life, except perhaps in sequential ways from the chronic form. There are many people with this infection who go through life with only slight disturbances and without being conscious of anything approaching invalidism. On the other hand, there are many who develop this condition, and definite states of ill health occur.

Culled from over two thousand records, the following associated conditions have appeared to be significant: "Acidosis", anxiety states, appendicitis, functional heart states, hypertrophic arthritis, "indigestion", pyloric and gastrospasm, proctitis, skin conditions, (persistent), toxic eye states and vasomotor disturb-

*_Editor's Note_:—This is the first of a series of four articles to be published on this subject. The second will be on putrefactive conditions, the third on the combined form and the last on specific infections of chronic types.

ances. In persons in whom the condition is well developed, a distinct invalidism exists and life may be considerably shortened in consequence. The most direful results are found, not in the pure states, but in those in which putrefactive states are also present.

The Patient

The individual is usually well nourished and overweight is common. Often in the well treated case, marked reduction in weight occurs without the employment of weight-reducing diets, and spastic states in the colon disappear without antispasmodic medications. Pyrosis and vague types of abdominal distress are compained of. Hypertension is common, which relieved through diet, suggests strongly that reduction diets in hypertension accomplish their results by semi-starvation (rice and weight-reduction diets) and by bringing about biochemical changes, the diets influencing the inimical bacteriology. The deep reflexes are increased. Gouty and transitory pains in various parts of the body are occasionally complained of.

The excessive production of ammonium butyrate, and organic fermentation acids with putrefaction products in mixed infections, is apt to produce irritable states of the digestive tract. There is a tendency toward desquamation of epithelium throughout the digestive tract including the mouth, and excessive cells and nuclei may be present in the stools. Patients who suffer from this condition do not tolerate carbohydrates and fats well. The significance of this is apparent in connection with weight-reduction diets both in obesity and the use of fat free diets in gallbladder disease. The benefits brought about by fat free diets that lower fatty acid formations have significance here in influencing the inimical bacterial state. Diarrheic spells may come from the excess of fatty acids. The disturbances are by no means limited to the digestive tract and often have importance in internal medicine. This has also been proven by animal experimentation. The nitrogen excretion becomes deficient. This with absorbed toxic substances, brings about metabolic disturbances in the liver and the highly specialized organs of the body. If the putrefaction is in excessive amounts, anemia may exist, sometimes even clinical findings of progressive pernicious anemia; this is probably due to absorbed hemolysine. Fatigue and loss of strength are common. The nervous state, gallbladder conditions and hypercholesteremia will be described later on.

To diagnose the condition properly, stool and urine specimens should be obtained by keeping the patient on the test diet advanced by the author[2], casual specimens being of no value. Leaving out the various matters pertaining to a proper and complete examination of stool and urine, the condition is suspected by the color of stools which is lighter than normal, sometimes being quite yellow or greenish. They are never condensed, float in water, are usually soft and mushy (these people are rarely constipated), and generally are interspersed with gas bubbles. The odor is sour rather than fetid, suggestive of the presence of organic acids (butyric, caporic propionic and valeric acids—the first two being the most abundant).

The general appearance of a Gram stained specimen of feces is characteristic in the condition. Free spores may be observed. The Gram negative organisms are low in number. When this is low as compared to the Gram-positives (which are mostly welchii and Gram positive cocci) diagnosis can be made. However, there are instances of definite acid fermentation in which the stool specimen does not show a predominance of the Gram positives (a normal stool is approximately one-third Gram positive to two-thirds Gram negative) and yet the clinical condition, the stool and urine examinations suggest the presence of this condition. Here, further examination of the stool is indicated. A highly aciduric stool may not be present, but there may be evidence of a putrefactive state (high indolic urine, Rosenback reaction and positive bichloride of mercury test). Here the anaerobes prepare the way for the breakdown of the protein substances in the bowel contents for the putrefactive organisms to finish, yet the anaerobes may be of fundamental significance. One then has to engage in phage studies of fluid medias to note the persistence of the anaerobes, and at times their identification, to note whether they are pathogenic, zymogenic or saprophytic, the pathogenic and zymogenic forms being of more importance than the saprophytic. These observations are made in aerogous, anaerogenous and strictly anaerobic ways which need not be gone into here. It is well to remember that the organisms of mischief are usually the most persistent and resistent to phagings, and should be studied daily for at least ten days. Much of bacterial significance can be learned from this method of study.

The urine, as a rule in the well established case, is generally free or very low in indican and ethereal sulphates. It is slightly higher than normal in specific gravity, rather highly colored, two to six times normal in acidity, and high in uroseine from carbohydrate break down (fermentation). The saliva may be markedly acid. In the cytology of urines evidence of kidney irritation may be present on microscopical examination. Hyaline casts, cylindroids and excessive epithelial cell shedding are seen.

Nervous States

It will become known eventually that not all functional nervous disorders are due to psychiatric conditions and only matters of personality, are of emotional and environmental origin. Metabolic and cytological cell changes causing function and structure defects are commonly developed as secondary states to internal conditions. In this connection, the biotoxic disorders, especially those of the intestinal tract, should receive a far greater interest on the part of the profession than exists today because it is commonly observed that correction or alleviation of these often brings about most pronounced psychiatric results.

The most common complaints are depressive mental symptoms often accompanied with general tension and irritable mental states. Various degrees of fatigue neurosis (neurasthenia) may be present and these may limit the person's ability and desire to work. Insomnia is common especially when putrefaction is also high. Sanitaria or long vacations may be required for relief of mental depressions. The enterogenic poisons may affect the motor structures of the central nervous

system (sometimes with the production of organic disease, progressive muscular atrophy, combined degeneration of the cord), the sensory paths (both peripheral and central), disturbances of the sympathetic nervous system and even the peripheral and central mechanisms that subserve the special senses. Before judging so-called psychotic disorders as simply those of personality, environmental, family and financial in cause, thorough investigation should be made of internal medical and especially chronic toxic disorders.

Hepatobiliary Tract Conditions

Various infective conditions have been proven to bring on organic disease of the liver (viruses, alcohol, syphilis, etc.). Cirrhosis probably is produced by alcohol in an indirect manner. Ordinarily the upper levels of the small intestine are sterile. In toxic gut conditions the bacteria of the lower ileum may mount up and ascend to those regions of the small intestine where the portal absorption is most active. When bacterial infections exist, toxic substances would be absorbed and carried to the liver by the portal blood. The toxic products absorbed, if not totally conjugated by the antitoxic powers of the liver, could in time produce organic disease and functional disturbances in the organ they immediately come in contact with. The common presence of hepatic cirrhosis in nonalcohol drinkers suggests that another factor, or factors, is important in its production, and it is of interest here that only the minority of heavy drinkers develop cirrhosis. Initiated by an anaerobic infection of the ileum and colon, the organisms of the anaerobic groups ascend in the small intestine and become established. Absorption of products from these bacteria in overwhelming amounts over long periods of time, and the effects of alcohol drinking on bacterial enhancement, is most probably the most common answer for cirrhotic liver states. To repeat, when such toxemias exist it is obvious that they can produce functional and organic disease in the liver.

There is good reason to believe that diseases of the biliary tract are initially caused by metabolic disorders in the liver and that the precipitation of bile salts and minerals in the production of cholelithiasis and gallbladder irritations from cholesterol are a result; thus the diagnosis and treatment of the common forms of gallbladder and duct states is fundamentally that of an intestinal disorder. Since this conception was put into practice, the author's results in handling gallbladder conditions has improved markedly[3], and duodenal drainages, cholagogues, etc., are today no longer used in treatment. The microorganisms met with in infected bile specimens and in examination of gallbladder walls are always of the intestinal forms, and in a series of 50 cases of chronic cholecystitis with and without stones, in which the bile contained organisms, the same bacteria were always isolated from the upper regions of the small intestine. When the liver is functionally disturbed and biliary infection exists, it may be too much to expect that treatment could produce substantial cure even though the patient claims definite clinical improvement. In the chronic gallbladder cases substantial results from intestinal treatment was the rule, unless stones were present giving clinical symptoms. In these cases surgery was necessary.

Hypercholesteremia

Cholesterol does not leave the body once it is formed, except in trivial amounts in feces. It is found in all animals and no doubt performs some function still not known. It is derived mostly from ingested foods (mainly fats) and no doubt is also formed in the body. It may be excessively accumulated in the body and may be deposited in the walls of the arteries with the production of sclerosis[4] or in some instances in the gallbladder with the formation of gallstones (80 per cent of gallstones contain cholesterol). It is also important in the production of chronic cholecystitis without stones and perhaps also in nephrosis. Since fatty acids are abundant in aciduric stools and hypercholesteremia is common in these toxemias, the importance of this intestinal condition in the production of excess amounts of cholesterol in the conditions mentioned is suggested. It no doubt can be easily oxyalized, as can be proven in diabetes by the use of insulin; by outdoor exercise and the taking of moderate amounts of alcohol. In the conditions mentioned, arteriosclerosis, gallbladder conditions, nephrosis and diabetes, low fat diets are advocated and have been proved of value in therapeutic ways. In this form of toxemia, fatty acids are manufactured in the intestine and readily absorbed. To limit the fat intake would be helpful, but for the logical control of these conditions the intestinal state is of greatest importance and of primary value, and thus these conditions should be attacked directly rather than be treated only by fat free diets and indirectly.

Treatment

In the order of importance the following is offered:

Biologic:—Considerable antagonistic actions exist between different strains of intestinal organisms, especially those of B. coli and streptococci. Nissle proved from bacterial observations that some strains of B. coli had a high antagonistic index and this was a valuable asset in the prevention of infection by pathogenic organisms. This antibiotic phenomena was proven by Gratia who showed that the antibiotic effect can exist even between different strains of B. coli itself. The same is true between certain of the anaerobes and coliform organisms. While this can be more or less depended upon in these acid fermentative conditions the best strain to employ is the B. coli, that is dextrose, lactose, methyl-red and salicin positive and saccharose negative. Other (stock) strains that have been efficient may be employed providing that they are studied by inoculation of the patient's stool with the stock strains or by those secured from others, and the phagings studied. At the beginning of treatment, the B. coli in the acid fermentation are low or at least inefficient in antagonist ways against the anaerobes. The fundamental step is to secure a strain of B. coli that is definitely antagonistic to the anaerobes present. This secured, 15 cc. of dextrose-peptone solution is inoculated, grown for 24 to 36 hours, made up to 160 cc. with 2 per cent lactose solution and injected into the rectum twice weekly. The B. coli used usually cannot be grown from the patient's stools at the start of treatment because if they were there in easily cultivatible amounts the anaerobes would not be active. In the course of a few

weeks, when the anaerobes have been reduced, auto-cultures serve the purpose during the rest of the treatment, which should be continued for several months. Such cultures act both directly and in a bacteriophagic way if grown autogenously from stools for two days. If one is correct in bacteriologic procedures (and constant checking of the stools should be done and perhaps shifts made with other strains of the same organism) one can expect results in four weeks time. After a satisfactory result has been accomplished, the treatments should be continued for at least thirty instillations. While this is by far the best type of treatment, stool examinations should be performed at monthly intervals for at least six months after the course of instillations to note the permanence of the bacteriologic state. Sometimes a permanent result is not secured, and short courses of biologic treatments are necessary at intervals for a year or more. This type of infection is usually easy enough to control, but for this to be continued in a permanent way is always questionable. Roughly, in about half of the cases the results are permanent, in about a quarter the anaerobes remain much reduced, and in about another quarter they tend to multiply and thus require more treatment.

Diet:—The diet should be of a high protein type in which three to ten per cent vegetables are used. Therefore, meats, fish, poultry, cheese, eggs, the legumens are the sheet anchor. No fats are allowed except a little fresh cream and butter occasionally. Milk is prohibited except that used in cooking. Sugary foods are not allowed. Since most of the intestinal organisms are factulative, dieting after a month is not necessary and the patient is then placed upon a normal plan in which the daily nitrogen intake is not above 90 grams (about 180 grams of high protein foods). Fats are then allowed. A list of foods in descending contents of protein follows:

The purpose of this diet is to increase the protein foods with limitation of the carbohydrates and fats. The foods especially to be recommended are listed below. Any food that disagrees can be eliminated. Some of the foods marked 1 2, 3 and 4 are to be eaten at each meal in the day and the more the better. The other foods are to balance the diet.

1. Beef, in any form
   Veal, in any form
   Lamb, in any form
   Pork, in any form
   Meat loaf
   Kidneys
   Liver
   Sweetbreads
   Turkey, dark meat
   Bologna
   Frankfurters
   Any fowl or game birds
   Tongue
   Tripe
   Brains
2. Blue fish, broiled or roasted
   Codfish, any form
   Halibut
   Mackerel
   Smelts
   Shad
   Clams
   Lobster
   Oysters
   Scallops
3. Buttermilk
   Koumiss
   Milk
   Brie cheese
   Cottage cheese
   Cream cheese
   Pineapple cheese
   Roquefort cheese
   Swiss cheese
   Butter
4. Eggs, any form (2)

   Asparagus
   Beans, lima
   Beans, string
   Peas
   Spinach
   Avocado, salad
   Celery salad
   Cheese and pineapple salad
   Cucumber salad
   Fruit salad

Mushrooms	Lettuce salad
Cabbage	Cole slaw salad
Celery	Pineapple salad
Tomatoes	Sardine salad
Rhubarb	Tomato jelly salad
Eggplant	Perfection salad
Soy beans	Watercress salad
Bouillon	Coffee jelly
Clam Chowder	Junket
Oyster stew	Lemon jelly
Vegetable soup	Orange-milk sherbert
Bean soup	Prune souffle
	Rice pudding cream
Brown sauce	Snow pudding
Cheese sauce	Sponge cake
	Salted nuts
No cereals	Raspberries (no sugar)
Boston brown bread	Apricots
Rye bread	Muskmelon
Corn muffins	Watermelon
Graham muffins	Blackberries (no sugar)
Griddle cakes	Orange
Bran muffins	Peaches (no sugar)
French toast	Jello
Saltines	
Soda crackers	Cocoa made with milk
Pretzels	Gingerale
Triscuit	Lemonade
	Egg Lemonade
	Soda water plain
	Tea

Fresh air:—Plenty of out-door exercise is advised. This includes all types of out-door games. Walking one to three miles a day is often satisfactory.

Biologicals:—The various nonabsorbable sulfa drugs and streptomycin have been tried many times without benefit in bringing about any substantial results, and usually without clinical benefit either. The same may be said of anaerobic vaccine of the gas bacillus type.

Drugs:—Instillations of free oxygen, as advanced by the author for the treatment of gout[5] (and since advised for intestinal conditions by Felsen), brings about considerable benefit, the instillations are about one liter in 24 hours. Whether this is accomplished by inhibiting the anaerobes with oxygen directly or indirectly by absorption is not known. It is usually not used except where bacteriologic treatment cannot be employed. The antacid drugs, both those that are readily absorbed and those that are more slowly so, occasionally serve for symptom relief and may be used for a short period in the beginning of treatment; calcium carbonate is best. The same is true of alkaline waters, but their price and inconvenience may make them impractical. They all depend upon the carbonate salts dissolved in them, and as good results are accomplished by a small amount of bicarbonate of soda in a glass of plain distilled water.

After the diagnosis of this intestinal state has been made, satisfactory worthwhile results in treatment can usually be depended upon.

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A REVIEW OF FIVE CASES OF PANCREATIC DISEASE*

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Introduction

The first comprehensive description of acute pancreatitis was contributed by R. H. Fitz[1] of Boston, in 1889. Since then many articles have appeared in the literature describing other less damaging pathologic conditions. Within the past year at Mountainside Hospital we have observed five different types of pancreatic disease which we are now reporting.

Calcification of the Pancreas

Francisco[2] in a review of the literature reported that up to the end of 1942, 18 cases had been reported. The diagnosis was usually made at autopsy but recently has been recognized through roentgen-ray study, or exploratory operation.

Case 1:—S. G. This 40-year-old male was first seen in 1946, at which time he was admitted to Mountainside Hospital complaining of acute, continuous, mid-epigastric pain, accompanied by severe vomiting. He admitted drinking heavily and noted about 20 lbs. loss of weight. A tentative diagnosis of ruptured peptic ulcer was made and he was explored on the following day. At operation the pancreas was found to be hard and calcarious, with induration. No free fluid or ulcers were found. Following the exploratory operation a serum amylase was reported as 676 mg. per 100 cc., Sugar 104 mg.; Lipase 1.4 units; urinalysis showed 3 plus albumen, negative sugar, a few RBC's and casts. Blood count was 4,770,000 RBC's, WBC's 8,500, Polys 90. Five weeks after operation his serum amylase was 78 mg. per 100 cc. Urine and blood count were normal; the glucose tolerance test showed slight elevation at three hours, 110 mg. Follow-up on 10/14/47—the serum amylase was found to be 520 mg. He did not appear acutely ill but was hospitalized for observation and diet routine. His glucose tolerance curve was normal. He has been followed in the gastroenterological clinic now for the past two years and only occasionally will he have pain in the epigastrium. This usually occurs after going off the diet or taking alcohol.

Carcinoma of the Pancreas

It has been estimated that from one to two per cent of all carcinomas occur in the pancreas. It is observed in .1 per cent of all patients admitted to hospitals. The delay in diagnosing pancreatic cancer is usually due to the difficulties encountered in examination of the organ, and to the unreliable results of the laboratory tests. Any patient over the age of forty, with a constant dull pain in the epigastrium, with loss of appetite and weight, should be suspected of having

*Observed at Mountainside Hospital, Montclair, N. J.

carcinoma of the pancreas. Jaundice may be a late sign and in many cases does not occur, as we have been led to believe from the old textbooks. A highly significant finding is the emotional disturbance in patients with pancreatic malignancy. This may be present in the form of anxiety, obstinate insomnia, and depression with crying spells. Many of these cases are usually dismissed as psychosomatics.

Case 2:—I. A. This 72-year-old female, ex-nurse and school teacher, was first seen on 2/17/48, complaining of a persistent distress under the left ribs for the past three months. There was loss of appetite and weight. The weight loss at the time was only 3 pounds, but to her this was significant because her weight had remained steady for many years. The pain in the left upper quadrant and slight pain in the epigastrium was intensified three hours after eating, but was relieved with alkalis. A gastrointestinal series failed to reveal any pathological process occurring in the stomach or duodenum. The gallbladder series showed good concentration of dye, but after a fatty meal failed to empty, and the pain was more severe. The sedimentation rate (Cutler) was 6 mm. in one hour, Serum amylase 60, and Lipase .5. The gastric analysis showed 7.5 degrees free HCl in one hour (Ewald).

Despite all treatments and diets she continued to have the dull, steady pain in the epigastrium, relieved partly by alkalis. Loss of weight and anorexia continued. She was finally referred back to her family physician with a diagnosis of chronic interstitial pancreatitis and poor functioning gallbladder. Five months later she was seen again in consultation and referred to the hospital for observation. There was now a 30 pound loss of weight and the pain was more intense. No masses were palpable in the abdomen and the point of tenderness was over the body of the pancreas. A gastrointestinal series was again reported as normal. With a Wagensteen drainage, and infusions, she had some relief of pain, and after a week of treatment food was re-instituted with alkalis. The pain returned. Her serum amylase during the acute attack was 379 mg. per 100 cc. blood, NPN 33, Chloride 511.5, Sedimentation rate (W) 55, Blood count showed WBC's 13,000, Polys 83. After treatment her serum amylase dropped to 73. She developed bronchial pneumonia which cleared after administration of penicillin. She gradually regained strength and was discharged from the hospital with a diagnosis of carcinoma of the pancreas. She expired one month later with general metastasis.

Acute Edematous Pancreatitis

Zoepffel[3] in 1922, reported four patients in whom, at operation, the pancreas was found to be swollen and edematous, but there was no evidence of pancreatic hemorrhage or necrosis. Elman[4], in an article in 1933, brought out the fact that this represented a form of acute inflammation which is less fulminating and is only transiently active. Elman stated that after adopting the amylase test, 65 cases of pancreatic disease were found in five years at the St. Louis Hospital, whereas previously there were only a few cases each year. Symptoms and signs such as shock, cyanosis, agonizing pain and muscle guarding are common findings.

Case 3:—A. T. This 34-year-old-female, two days prior to attack of extreme shock, developed an upper respiratory infection which she treated with a few alcoholic drinks. The day before admission to the hospital she had nausea and vomiting, felt weak, and had vertigo. There was only slight epigastric pain following the vomiting. That evening she went into shock and was admitted to the hospital. Past history revealed no serious illnesses. She had been taking alcohol in larger amounts lately. She had had some anorexia and loss of weight in the past three months and had been given iron and liver shots by her physician. On admission to the hospital she was dyspneic and comatosed. Extremities were cold; pulse weak, about 130. Dehydration was marked, tongue dry and coated brown. There was no rigidity of the neck or petechiae of the skin. The heart sounds were distant and faint. The B/P was 30/0. There was slight tenderness in the epigastrium without rebound tenderness, and general spasm over all the abdomen. There was no doubt that this patient was in extreme shock. The chest and abdominal x-rays were negative. An infusion of 1,500 cc. of 5 per cent glucose and saline was given in the sternal marrow because of the collapsed condition of the blood vessels. Coramine and adrenal extract were also given. Laboratory tests showed 39 per cent Hemoglobin, RBC's 2,800,000, with 10,000 WBC's and 90 per cent Polys. Plasma through the sternal marrow was also given. Within the hour the blood pressure improved and blood was able to be given through the vein. Slight tenderness and rigidity over the pancreas were noted. A serum amylase was 320 mg. per 100 cc., the NPN was 65, Sugar 154, Chlorides 577. There were no sickling of RBC's and she had a negative Kline. Two days after admission a temperature of 103 with rales in the chest and consolidation, were noted. This was treated with penicillin. The blood picture improved with transfusions; Wagensteen drainage and alkalis improved the gastrointestinal complaints, and in one week this patient was improved enough to take a bland diet. The serum amylase was 130, NPN 44, Sugar 119. Gastrointestinal and gallbladder series were negative. Barium colon x-rays and pyelograms were also negative. The last serum amylase before discharge was 81. All liver tests were negative.

Adenoma of the Pancreas

Benign tumors of the pancreas are rare and the estimated frequency ratio is one nesidioblastoma in 800 autopsy subjects. Only 20 per cent of those afflicted, however, display symptoms, so that the vast majority of nesidioblastomas are purely incidental findings (Whipple and Frantz[5]). A recent survey of Sproul[6], described twenty-five instances of benign epithelial tumors of the pancreas in 4,258 consecutive autopsies.

Case 4:—J. G. This 47-year-old male, for four years prior to his last admission had been a patient in this hospital for various complaints ranging from alcoholism to psychoneurosis. On one occasion he was admitted in a semicomatose condition following an auto accident. A fracture of the skull was ruled out by x-ray. On other occasions he complained of headaches, and nausea and vomiting, with general weakness. All tests were negative except on one occasion the blood sugar was 34 per cent, but on the next admission the blood sugar was 62 per cent.

This patient was seen many times in the outpatient department, always with the same complaint of vague abdominal pain; nausea and vomiting; weakness and fainting; mild frontal headaches. He noted that after eating he would feel better and carried a sandwich with him to prevent these attacks. Finally, on the last visit, another blood sugar was done and showed 20 per cent glucose. He was admitted and came out of the semicomatose condition with 50 per cent glucose given intravenously. An adenoma of the pancreas was then suspected. All blood tests were normal except the glucose tolerance curve. It was—fasting 14, 1 hr. 91, 1½ hr. 119, 2 hr. 73, 3 hr. 53, and repeated, showed the same picture. A gastrointestinal series revealed the presence of a small ulcer on the lesser curvature of the stomach. He was transferred to Surgery, and at the operation (performed by Dr. Moeckel), an islet cell adenoma was found in the portion of the pancreas removed. It was a small, firm nodule, measuring 1.3 x 1.0 x 1.0 cm. This was diagnosed pathologically as islet cell adenoma. The gastric ulcer was not disturbed but the spleen was removed to aid in removing the pancreas. The patient made a good recovery and the blood sugars were followed postoperatively. They rose to 133, 194, 150, 189, 159, 123, 104 mg. The urine for seven days showed sugar but remained clear afterwards. Another glucose tolerance test was done and showed—fasting 94, 30 min. 159, 1 hr. 238, 1½ hr. 212, 2 hr. 212, 3 hr. 194. Gastrointestinal x-ray showed a healing gastric ulcer. Patient has remained well for the past eight months.

Pancreatic Injury

In 1930, E. L. Stern[7] described traumatic injuries to the pancreas and stated that these were most often due to operative procedures such as subtotal gastric resection for ulcer of the duodenum, cholecystectomy, incision for pancreatic calculi and diagnostic biopsy of the pancreas and others. The most frequent occurred after stomach operations, according to the listing given by Schmieden and Sebening[8]. Acute pancreatic necrosis is the most common type of pancreatic lesion induced by operative procedure.

Case 5:—G. S. This 60-year-old white male gave a history of chronic duodenal ulcer for many years, and prior to admission to the hospital had been in almost constant pain, with nausea and vomiting for two weeks. He responded to treatment in the hospital for the first week but when solid food was tried, the pains returned and operation was advised. A gastrointestinal series revealed the presence of a penetrating ulcer, probably into the pancreas.

At operation a deep penetrating ulcer of the wall of the duodenum was found to be imbedded into the pancreas and had ruptured into it. A gastric resection was done by Dr. V. Seidler and the ulcer freed from the pancreas and sewed in. This patient made a good recovery for the next five days but on the fifth day developed signs of peritonitis and acute pain over the pancreatic region. Bloody serum material in large amounts oozed from the drainage wound. The patient's pain was not relieved with large doses of morphine and he quickly went into shock, cyanosis and clinical features of severe hemorrhage. The patient expired soon after the initial pain and shock despite infusions, etc.

SUMMARY

1. Five different cases of pancreatic disease are presented and described briefly to emphasize early diagnosis.

2. Serum amylase blood test was of great value in the diagnosis.

3. Every acute case of upper abdominal pain admitted to the hospital should routinely have a serum amylase blood test.

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DISTASTE FOR SMOKING: AN EARLY SYMPTOM IN VIRUS HEPATITIS*

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During the past year I have noted, with some consistency, a previously undescribed symptom in the early stage of acute virus hepatitis, namely, a distaste for smoking of tobacco where the patient was previously a regular smoker. This distaste for smoking is fairly abrupt in onset. It is associated with an altered unpleasant taste, where smoking has previously been a source of comfort and pleasure to the patient. Some patients have described a "musty taste", others a "bad taste". I wish to make it clear that the symptom to which I refer differs from the rather common reaction on the part of many smokers who may cease their smoking when they develop "colds" or sore throats or any respiratory infection. In the latter instance, close questioning will usually reveal that the reason for ceasing to smoke is either an irritation of a sore throat or a decreased ability to taste or smell the tobacco, not a newly acquired positive distaste of an unpleasant nature.

This symptom was first brought to my attention when I was consulted by M. C., a 24-year old physician, complaining for three days of fever, malaise, anorexia, nausea and abdominal distress. Unsolicited, he stated that abruptly with the onset of his illness he had developed a distaste for cigarettes, whereas previously he customarily smoked heavily. Examination revealed no jaundice, low grade fever, bilaterally enlarged posterior cervical lymph nodes, the liver palpable at the costal margin and delayed tenderness over the liver on fist percussion. On the eighth day of illness, his thymol turbidity test was elevated to 13.6 units, alkaline phosphatase to 7.0 units. A single cephalin flocculation test, performed on the first day of symptoms, was 2+ at forty-eight hours. He recovered gradually over a two-week period, remaining unjaundiced. He resumed cigarette smoking about the same time that his appetite returned to normal.

Since then I have included a question about smoking habit in taking a history from all cases suspected of virus hepatitis. I have encountered a total of twenty-one cases, of which ten were nonsmokers. Among the eleven smokers, a definite complaint regarding smoking distaste was clearly elicited from eight, was absent in one and was equivocal in two. A brief description of each of these eleven cases will be given. The first was the original patient mentioned previously.

The second was M. L., a 41-year-old female, with icteric hepatitis of the serum-transmitted variety. She readily admitted to having ceased smoking cigarettes completely (her usual quota had been four packages per week) because of a sudden distaste for them about ten days before jaundice was noted.

The third case was that of D. S., a 41-year old male, with sporadic anicteric hepatitis of a mild type. His usual quota of cigarettes was six per day. Upon first

*From the Medical Service, Beth Israel Hospital.

presenting himself, he complained of nonspecific symptoms such as weakness, generalized aches and headaches. Unsolicited, he then remarked that he was amazed at his recently acquired unpleasant taste upon smoking, sufficiently annoying to have caused him to stop smoking completely for the past three days. His appetite was relatively undisturbed. My awareness of this smoking complaint led to a work-up with hepatitis in mind. His liver was not palpable, but it was minimally tender on fist percussion. His serum thymol turbidity was elevated to 8.4 units.

The fourth patient, A. C., a 29-year old merchant seaman, with sporadic icteric virus hepatitis, had a somewhat different reason for eliminating smoking. Ordinarily, he consumed a package of cigarettes daily, but with the onset of his epigastric distress, jaundice and anorexia, and while still ambulatory, he discovered that smoking aggravated his abdominal symptoms. Whereas, in the previous three cases, the unpleasantness was a positive one and was related to taste and referred to the mouth, in this instance it was clearly related to an exacerbation of abdominal discomfort and vaguely associated with distaste.

The fifth case was an instance of very severe icteric hepatitis in I. S., a 38-year old surgeon. The mode of his infection may possibly have been accidental pricking of his fingers during the course of intricate thoracic surgical operations. For four weeks before he became jaundiced, he complained of nebulous symptoms, but predominant were violent distaste and nausea on smoking cigars. Ordinarily, he smoked four cigars daily. During the entire period of his illness these symptoms persisted. Even smoking by others (including cigarette smoking) in his presence caused him similar distaste and nausea. He was jaundiced for three months. At the time of the present writing, 12 months later, his cigars taste "different" and are not so enjoyable to him as they used to be.

The sixth patient, H. G., was a 49-year old foreign military man with icteric hepatitis acquired by blood transfusion. He habitually smoked ten to twelve cigarettes per day. Whenever he developed an illness he was in the habit of stopping his smoking. He therefore ceased smoking completely after sustaining severe injuries in an automobile accident. A month later, he received the blood transfusion and three months after that he became jaundiced. During the few weeks before jaundice became apparent, while he felt weak, febrile and was losing his appetite, he attempted to resume smoking. He could not tolerate the cigarettes because of a definite "mal goût" (the interviews took place in French). This "mal goût" disappeared during his convalescence.

Case seven is that of H. F., a 69-year old male, with anicteric hepatitis, whose outstanding complaints were asthenia, loss of appetite, pain in the upper abdomen and lower anterior chest, and an abrupt onset of "musty taste" on smoking cigarettes. His usual daily quota of cigarettes was seven, but for a seven to eight weeks' period he could not tolerate their taste. His liver was enlarged and tender for several weeks; his thymol turbidity rose to 8.6 units, cephalin flocculation test to 2-3+.

Case eight, that of A. G., a 61-year old male, is available to me through the cooperation and courtesy of Dr. Zachary H. Benjamin, for consideration only in a retrospective sense. Dr. Benjamin had already published the report of this case as one of hepatitis due to cincophen, with recovery[1], when he became aware of my interest in smoking distaste. He then remembered that one week before the onset of any other symptoms (and at least two weeks before jaundice set in) his patient had ceased smoking cigarettes because of a positive distaste which he had suddenly acquired for them. Ordinarily, he had smoked a package daily. During the six weeks of acute illness, he could not tolerate smoking by others about him, nor even the odor of a stale, unlighted cigar. Clinically, this patient followed closely the pattern of cholangiolitic hepatitis as described at length by Watson and Hoffbauer[2] in 1946. Because of the considerable evidence in favor of an obstructive type of jaundice, he was explored surgically but no obstruction of the extrahepatic biliary tract was found. A biopsy of the liver taken at operation showed central bile stasis, swelling of liver cells with granular degeneration and binucleated liver cells. There was a history of recent cincophen ingestion. Nonetheless, I believe this entire picture, including the biopsy, could be that of cholangiolitic hepatitis.

I shall now describe the two cases with "equivocal" findings. The first patient was M. C., a 57-year old male, with cholangiolitic hepatitis. He was deeply jaundiced, and, like the case of A. G. above, was explored surgically. No extrahepatic biliary obstruction was found. Liver biopsy revealed only central bile stasis and fatty change. He gradually improved. He ordinarily smoked one cigarette per day. This in itself makes it difficult to evaluate his reaction to smoking during his illness, and it might have been wiser to include him among the nonsmokers. However, early in his illness he did tell me, on questioning, that he recalled no change in taste on smoking his cigarette, but had ceased smoking with the first signs of jaundice three weeks previously, merely because he was ill. A few days later, he recalled to another physician that when he had last smoked, the cigarette had tasted peculiarly and that was a contributing factor in his stopping smoking.

The second equivocal case was that of H. G., a 27-year old male, with mild sporadic anicteric hepatitis. He usually smoked a package of cigarettes daily, but reduced this to five per day at the onset of his illness because of lack of taste rather than unpleasant taste. However, for two days after his temperature became normal, smoking resulted in an unpleasant taste, nausea and vomiting.

The "negative" case was that of L. S., a 48-year old male, with icteric sporadic hepatitis. He was a heavy cigar smoker who noted no distaste during his illness, although he did stop for several days because his throat was irritated.

With but one possible exception, which I shall describe shortly, in no other instances of hepatitis or diseases commonly associated with hepatic involvement[3, 4, 5, 6], have I encountered this symptom. I have looked for it unsuccessfully in ten cases of infectious mononucleosis, six cases of virus pneumonia, three

cases of cirrhosis of the liver, one case of subacute bacterial endocarditis, one case of chronic hepatitis and one case of metastatic adenocarcinoma of the liver. Also it was lacking in one case of obstructive jaundice due to carcinoma of the gallbladder and in two cases of stones in the common bile duct.

The single possible exception to which I alluded above is an interesting case, which I am reporting elsewhere in full[7], of chronic hepatitis with cirrhosis following infectious mononucleosis. I have had the opportunity to study this man over several years and during several hospital admissions. His first admission, in 1945 at the age of 25, was for an acute illness with jaundice, epigastric and right upper abdominal pains, epistaxis, vomiting and loss of weight. He had hepatic enlargement and tenderness, and liver dysfunction was corroborated by chemical examinations. The final diagnosis was infectious mononucleosis. This diagnosis was abetted considerably by a sheep cell agglutination titer of 1:800. On his sixteenth hospital day he signed himself out of the hospital but returned four days later with an exacerbation of his abdominal distress. During his subsequent stay he admitted that smoking cigarettes had aggravated his abdominal pain and continued to do so. Admittedly, it is not clear how much the premature ambulation alone was at fault.

Through the generous cooperation of Dr. John R. Neefe of Philadelphia, recently a rapid survey was instituted among thirty-three patients with acute hepatitis, to determine the incidence of smoking distaste. The following is the result of this survey:

"Of the thirty-three, twenty-one noted loss of taste for tobacco. Of these, many stopped smoking completely for a short period of time. All cut down in their consumption or discarded their cigarettes after only a few puffs. Some attempted a change in brand in order to overcome their distaste for cigarettes. Twelve of the thirty-three noted no change in taste for tobacco and smoked as much as usual despite anorexia and other symptoms."[8]

Discussion

Among the eleven patients with acute hepatitis whose cases I have described, eight, or 73 per cent, manifested a clear complaint of distaste for smoking. These eight patients included one cigar smoker and seven cigarette smokers. In one of these eight patients the smoking resulted more emphatically in an aggravation of abdominal symptoms rather than in distaste. The data obtained from the survey of thirty-three patients by Neefe and Shaffer revealed a distaste for tobacco in twenty-one (64 per cent). Adding both groups of cases together yields an incidence of two in every three cases, 66 per cent, thus:

Reported By	Total No. Patients (All Smokers)	Patients Manifesting Distaste for Tobacco	
		No.	%
Leibowitz	11	8	73
Neefe & Shaffer	33	21	64
TOTAL	44	29	66

I have no knowledge of the incidence of smokers among patients with acute hepatitis, but undoubtedly it varies in different groups of patients. In a military population, for example, it is doubtless high, and the number excluded from a study of this symptom would probably be smaller than in a segment of the population at large such as my study represents. Among my 21 cases, ten (48 per cent) were nonsmokers, but this number is relatively high, probably because it includes five cases of elderly women with serum-transmitted hepatitis. However, if the incidence of distaste for smoking should remain at two-thirds of virus hepatitis cases, even only among those who use tobacco, it is still a considerable percentage and, therefore, this symptom is worth seeking in eliciting a history.

I can offer no explanation for this interesting symptom. My observations have persuaded me to look for hepatic involvement where it is elicited, but this is by no means a proven point. If it is related to liver involvement, it may prove to be more generally indicative of liver parenchymal involvement of varied causes rather than rigidly reflecting virus hepatitis of the infectious hepatitis (I.H.) or serum-transmitted (S.H.) varieties, using the terminology suggested by Neefe[9]. Many more observations and accumulated experiences are needed before any legitimate conclusions in this regard may be drawn.

An investigation which immediately suggests itself is its relation to the pregnant state, because this represents the only other instance wherein I have encountered an exactly similar complaint with respect to smoking. Such a study I am now undertaking. The first pregnant lady studied because of this distaste for smoking cigarettes had a 3+ cephalin flocculation test and a thymol turbidity of 6.7 units.

Summary

1. A hitherto undescribed early symptom in virus hepatitis, distaste for smoking, is described in 8 of 11 cases.

2. No explanation for this symptom is offered but a relationship to hepatic involvement is suggested.

Addendum

Since the time this paper was originally written and submitted for publication, the author has studied an additional 20 cases of acute virus hepatitis. Of these 20, 7 were non-smokers. Of the remaining 13 patients, 8 gave a positive history for tobacco distaste (7 to cigarettes and 1 to cigars), 2 were negative and 3 were equivocal. Thus, the incidence of smoking distaste among the 24 smokers in the author's series of 41 cases is 66 per cent.

Acknowledgement with thanks is made to Dr. John R. Neefe for his generous cooperation and helpful advice; to Drs. Hyman Bakst, Zachary Benjamin, Leo Davidoff, Louis Greenwald, Milton Kramer, Joseph Litwins and Gary Zucker for permission to present data in their cases; and to Virginia Rechnitzer, Naomi Schecter and Daria Pikula for their technical assistance.

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EDITORIAL

Forgotten Names: Errors of Priority Credit In Medicine

"But these are deeds which should not pass away, And names that must not wither"—Byron.

When cultured, learned and long experienced physicians give thought to the workers of the past—to those who have, against many obstacles, and with almost no facilities whatever, contributed so much in the earlier years of medicine, to aid those of us who in the more recent decades have made "new discoveries" and "new inventions" and described "new diseases" and "new signs"—we must all agree that there are many notable names in medicine and surgery and in other fields of research, that *must not be permitted to wither, die and be forgotten!*

Patrick Henry, on March 23, 1775, at the Convention of Delegates of Virginia, among other things said: "I have but one lamp, by which my feet are guided and that is the lamp of experience". Also, "I know of no way of judging of the future but by the past".

Luther R. Marsh in "The Past and the Future"—"Would not backward roll the tide of time,

Though freighted, rich, with golden memories,
With large experience, and with hosts of friends,
The past is past, and cannot come again,
Sweet as it was, and laden with all joys—
Each day a pleasure and each morn a hope—
Yet it is fruitless to recount those scenes.
The wise men of the past, each in his way,
Gave out the wisdom fitted for his time.
But we have sailed away, far out of sight
Of all their maxims and their sage conundrums
The rising sun we need, to flood his light
Upon our pathway through the vast unknown
Why pore we o'er the history of time gone,
When our work lies in time that is to come?
Buckle we on for the advancing years;
Not ruminate on deeds by others done."—

May I suggest that Jessie Dobson's "Anatomical Eponyms", (London, 1946) be read—especially, "Eponymous Nomenculature" pages 1-13. Also, of interest, are: Hamilton Bailey and W. J. Bishop's "Notable Names in Medicine and Surgery" (London, 1946) and "Discoverers of Medicine" by William H. Woglom of Teaneck, N. J., (New Haven and New York, 1949), and "Harvey and Cesalpino's Role in the History of Medicine" by Sigismund Peller of New York (Bulletin of the History of Medicine, **23**:213-235, May-June 1949).

How many of us recall Matteuci of Pisa, who in 1843, one hundred and six years ago, knew that electric currents were produced by cardiac contractions, as

did also Kölliker and Müller in 1856? How many of us recall that quinidine was first isolated by van Heijningen in 1849, and by the great Pasteur in 1853? It was W. von Frey who found that quinidine gave such good results in flutter and auricular fibrillation!

Quinine was found, by Karel F. Wenckebach of Vienna, in 1914, to restore normal rhythm in auricular fibrillation. Very few modern physicians know of Leonard Fuchs (Fucius), the German botanist, who, in 1542, gave us the name *digitalis* for Withering's (1785) foxglove.

In the 13th or 12th century B.C. a monograph appeared on "Remedies for Diseases of the Anus". There even was a rectal specialist known as the "Guardian or Shepherd of the Anus". It is probable that the text of this early monograph is considerably older! (Bull. Hist. Med. **22**:311, May-June 1949).

It was Aristotle who gave us the name aorta, for the great blood vessel.

According to George Eliot, Silas Marner (1861), inherited from his mother some acquaintance with medical plants. He searched the fields for foxglove and other healing herbs. One day he saw his shoe cobbler's wife seated by the fire and suffering from heart disease and dropsy that similarly had preceded his mother's death. His mother, having experienced relief from a simple preparation of foxglove, he promised Sally Oates something to ease her distress since her local doctor was unable to help her!

There have been many young eminent undergraduates who contributed much of value to medicine and I suggest that "Eminent Undergraduate Observers" be read (Medical Life, N. Y. **32**:111-116, 1925). Another article of interest is James F. Brailsford's "Roentgen's Discovery of X-rays" (J. Internat. Coll. Surg. **9**:694-704; page 729, Nov.-Dec. 1946), as is also "Shakespeare's Medical and Surgical Knowledge" by John W. Wainwright, (New York, 1915).

"*Facts Are Stubborn Things*" (Eliot). HYMAN I. GOLDSTEIN

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New York, N. Y.

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Phoenicia, N. Y.

SAMUEL WEISS, M.D.
New York, N. Y.

PROGRAM COMMITTEE

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CHARLES W. McCLURE, M.D.
Boston, Mass.

LADIES PROGRAM COMMITTEE

MRS. FRANCIS T. JANTZEN — CO-CHAIRMEN — MRS. ANTHONY BASSLER

REGISTRATION—All members and guests should register. Identification badges for admittance to meetings will be given to those who register. These should be worn at all times during the session. Registration will take place at the registration desk on the convention floor.

LADIES REGISTRATION—At the registration desk on the Convention Floor. Registration facilities will be open at 8:30 each morning. Information concerning the various activities and events will be available there.

MEETINGS are held on Daylight Saving Time and will begin promptly at the time specified.

COURSE IN GASTROINTESTINAL SURGERY—Admittance only upon presentation of official matriculation card.

TECHNICAL EXHIBITS under the direction of Mr. Steven K. Herlitz, Exhibit Manager, will be open on Monday, Tuesday and Wednesday.

Those attending the Convention are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with the many new products and new equipment on display.

VISIT THE EXHIBITS

Program

FOURTEENTH ANNUAL CONVENTION
NATIONAL GASTROENTEROLOGICAL ASSOCIATION

SCIENTIFIC SESSIONS

24, 25, 26 OCTOBER 1949

HOTEL SOMERSET

Boston, Mass.

Members of our Association and subscribers to THE REVIEW OF GASTROENTEROLOGY, whether present or not at the time of the reading of a paper are always looking forward to its being published, so that they may read it carefully. With that in mind the essayists have been asked to submit their papers for publication.

FIRST SESSION

MONDAY MORNING, 24 OCTOBER 1949

WILLIAM REID MORRISON, A.B., M.D., F.A.C.S., President, National Gastroenterological Association, Chairman.

ANTHONY BASSLER, M.D., F.A.C.P., LL.D., Honorary President, National Gastroenterological Association, Co-chairman.

9:00 A.M.

1. **"Current Trends in Selection of Patients for Surgery in Duodenal Ulcer".**

Speaker

FRANCIS D. MOORE, M.D., Boston, Mass., Moseley Professor of Surgery, Harvard Medical School, Surgeon-in-Chief, Peter Bent Brigham Hospital.

9:30 A.M.

Discussers

JOHN W. SPELLMAN, M.D., Boston, Mass., Surgeon-in-Chief, St. Elizabeth's Hospital.

FELIX CUNHA, M.D., San Francisco, Calif.

DONALD C. COLLINS, M.D., Hollywood, Calif.

General Discussion

10:00 A.M.

2. **"The Diagnosis and Management of Splenomegaly".**

Speaker

WILLIAM B. CASTLE, M.D., Boston, Mass., Professor of Medicine, Harvard University; Director, Thorndike Memorial Laboratory; Director, Second and Fourth Medical Services, Boston City Hospital.

VISIT THE EXHIBITS

10:30 A.M.

Discussers

FERNANDO MILANES, M.D., Havana, Cuba
EDWARD A. COONEY, M.D., Boston, Mass.
WILLIAM C. MALONEY, M.D., Boston, Mass.

General Discussion

11:00 A.M. Recess to visit the Commercial and Technical Exhibits.

11:20 A.M.

3. "Experientia Docet".

Speaker

RT. HON. THE LORD ALFRED WEBB-JOHNSON, London, England, M.B.; Ch.B; F.R.C.S.; Hon. LL.D.; Hon. F.A.C.S.; Hon. F.R.C.S.I.; Hon. F.R.C.S.E.; Hon. F.R.A.C.S.; Hon. F.F.R.P.S.; Knight Commander of the Victorian Order; Commander of the British Empire; Distinguished Service Order; Territorial Decoration; Deputy Lieutenant of the County of London; Doctor of Laws; President of the Royal College of Surgeons of London, England.

11:50 A.M.

Discussers

ARTHUR W. ALLEN, M.D., Boston, Mass., Past President, American College of Surgeons.
WILLIAM W. LERMANN, M.D., Pittsburgh, Pa.

General Discussion

12:30 P.M.

LUNCHEON AND ROUND-TABLE DISCUSSION.

Subject

"Errors of Priority Credit in Gastroenterology and Cardiology".

Speaker

HYMAN I. GOLDSTEIN, M.D., Camden, N. J., Historian, National Gastroenterological Association.

SECOND SESSION

MONDAY AFTERNOON, 24 OCTOBER 1949.

HALSEY B. LODER, M.D., President, Boston Chapter, National Gastroenterological Association, Chairman.
DWIGHT O'HARA, M.D., Dean, Tufts College Medical School, Co-chairman.

2:00 P.M.

4. "The Problem of Gallbladder Disease".

Speaker

ALBERT F. R. ANDRESEN, M.D., Brooklyn, N. Y., Professor of Clinical Medicine, Long Island College of Medicine; Attending Physician (Gastroenterology), Long Island College Hospital.

VISIT THE EXHIBITS

2:30 P.M.

Discussers

HORACE W. SOPER, M.D., St. Louis, Mo.
ANTHONY BASSLER, M.D., New York, N. Y.
ROWLAND RICKETTS, M.D., Merchantville, N. J.

General Discussion

3:00 P.M.

5. "Transthoracic Gastric Surgery".

Speaker

RICHARD H. SWEET, M.D., Boston, Mass., Associate Clinical Professor, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital.

3:30 P.M.

Discussers

EDWARD D. CHURCHILL, M.D., Boston, Mass., Professor of Surgery, Harvard Medical School; Surgeon-in-Chief, Massachusetts General Hospital.
JOHN STRIEDER, M.D., Brookline, Mass.

General Discussion

4:00 P.M. Recess to visit the Commercial and Technical Exhibits.

4:20 P.M.

6. "The Significance of the Gastrointestinal Tract in Emotional Maturation".

Speaker

HENRY M. FOX, B.S., M.D., Boston, Mass., Assistant Professor of Psychiatry, Harvard Medical School; Senior Associate in Psychiatry, Peter Bent Brigham Hospital.

4:50 P.M.

Discussers

JEROME S. LEVY, M.D., Little Rock, Ark.
MERRILL MOORE, M.D., Boston, Mass.

General Discussion

5:30 P.M.

ANNUAL MEETING OF THE ASSOCIATION—GENERAL ASSEMBLY.

6:00 P.M.

CONVOCATION. Presentation of Fellowship Certificates.

ADOLF ABRAHAM, M.D., New York, N. Y.
IRVING A. BECK, M.D., Providence, R. I.
ALEXANDER J. A. CAMPBELL, M.D., Boston, Mass.
DONALD C. COLLINS, M.D., Hollywood, Calif.
JOHN H. CRANDON, M.D., Boston, Mass.
DANIEL DIAMOND, M.D., Brooklyn, N. Y.
D. JOSEPH DUGGAN, M.D., Malden, Mass.
BERNARD J. FICARRA, M.D., Brooklyn, N. Y.
HYMAN FISHER, M.D., Sunmount, N. Y.
PHILIP S. FOISIE, M.D., Boston, Mass.
MARTIN FRIEDRICH, M.D., Brooklyn, N. Y.
HERBERT GREENFIELD, M.D., Newark, N. J.
JACK GREENFIELD, M.D., Memphis, Tenn.
DWIGHT E. HARKEN, M.D., Boston, Mass.
JOHN DAVIS HUGHES, M.D., Memphis, Tenn.

Francis T. Jantzen, M.D., Boston, Mass.
Donald S. Jurnove, M.D., New York, N. Y.
William Leet, M.D., Providence, R. I.
Jean LeSage, M.D., Montreal, Canada
John H. McGowan, M.D., Quincy, Mass.
Louis H. Nason, M.D., Boston, Mass.
Carl H. Rabin, M.D., New Orleans, La.
Max Ritvo, M.D., Boston, Mass.
Nathaniel Edw. Rosset, M.D., Memphis, Tenn.
Harry Welson Rothman, M.D., New York, N. Y.
Max Harold Ruby, M.D., Waterbury, Conn.
R. L. Sanders, M.D., Memphis, Tenn.
Michael Scimeca, M.D., Brooklyn, N. Y.
Joseph Shaiken, M.D., Milwaukee, Wisc.
Samuel Sober, M.D., New York, N. Y.
Joseph Tartakoff, M.D., Boston, Mass.
Leonard D. Williams, M.D., Plainsfield, N. J.

6:30 P.M.
PRESIDENT'S ANNUAL RECEPTION (*Admission by card only*).

THIRD SESSION

TUESDAY MORNING, 25 OCTOBER 1949.

Frank A. Cummings, M.D., President, Rhode Island Chapter, National Gastroenterological Association, Chairman.

Jesse P. Eddy, III, M.D., Secretary, Rhode Island Chapter, National Gastroenterological Association, Co-chairman.

9:00 A.M.

7. "The Present Status of the Treatment of Cirrhosis of the Liver".

Speaker

Chester S. Keefer, B.S., M.S., M.D., D.Sc., Boston, Mass., Wade Professor of Medicine, Boston University School of Medicine; Physician-in-Chief, Massachusetts Memorial Hospitals.

9:30 A.M.

Discussers

Fernando Biguria, M.D., Brookline, Mass.
Charles S. Davidson, M.D., Boston, Mass.

General Discussion

10:00 A.M.

8. "Surgical Treatment of Duodenal Ulcer. A Comparison of the Results of Treatment With and Without Vagotomy".

Speaker

George Crile, Jr., Ph.B., M.D., Cleveland, Ohio, Member of Staff, Department of Surgery, Cleveland Clinic Hospital.

10:30 A.M.

Discussers

James T. Nix, M.D., New Orleans, La.
Carl Bearse, M.D., Boston, Mass.
M. E. Steinberg, M.D., Portland, Ore.

General Discussion

11:00 A.M. Recess to visit the Commercial and Technical Exhibits.

VISIT THE EXHIBITS

11:20 A.M.
9. "Ulcerative Colitis".
Speaker
FRANK H. LAHEY, M.D., Boston, Mass. Director, Lahey Clinic; Surgeon, New England Deaconess and New England Baptist Hospitals.

11:50 A.M.
Discussers
WILLIAM E. BROWNE, M.D., Boston, Mass., Surgeon-in-Chief, Carney Hospital.
SAUL SCHAPIRO, M.D., Brooklyn, N. Y.
EDWARD T. WHITNEY, M.D., Boston, Mass.
General Discussion

FOURTH SESSION

TUESDAY AFTERNOON, 25 OCTOBER 1949.

1:00 P.M.
MEETING OF THE NATIONAL COUNCIL.
WILLIAM C. JACOBSON, M.D., President, New York Chapter, National Gastroenterological Association, Chairman.
JAMES M. FAULKNER, M.D., Dean, Boston University School of Medicine, Co-chairman.

2:00 P.M.
10. "Traumatic Injuries to the Liver".
Speaker
GEORGE W. PAPEN, M.D., F.A.C.S., Boston, Mass., Surgeon-in-Chief, First Surgical Service, Boston City Hospital and STANLEY MIKAL, M.D., A.B., Boston, Mass., Teaching Resident Surgeon, First Surgical Service, Boston City Hospital.

2:30 P.M.
Discussers
LESTER R. WHITAKER, M.D., Portsmouth, N. H.
HENRY E. GRODEN, M.D., Boston, Mass.
General Discussion

3:00 P.M.
11. "Radiology of the Esophagus".
Speaker
RICHARD SCHATZKI, M.D., Boston, Mass. Instructor in Radiology, Harvard Medical School; Chief Radiologist, Mount Auburn Hospital, Cambridge.

3:30 P.M.
Discussers
FRANK J. BORRELLI, M.D., New York, N. Y.
FRANZ J. LUST, M.D., New York, N. Y.
LOUIS L. PERKEL, M.D., Jersey City, N. J.
General Discussion

4:00 P.M. Recess to visit the Commercial and Technical Exhibits.

VISIT THE EXHIBITS

4:30 P.M.

12. "Lesions of the Esophagus".

Speaker

DWIGHT E. HARKEN, A.B., M.D., Boston, Mass., Assistant Clinical Professor of Surgery, Harvard Medical School; Visiting Surgeon for Thoracic Surgery, Boston City Hospital; Senior Associate in Thoracic Surgery, Peter Bent Brigham Hospital; Thoracic Surgeon, Mount Auburn Hospital, Cambridge; Thoracic Surgeon, Malden Hospital, Malden; Thoracic Surgeon, Waltham Hospital, Waltham; Consultant in Thoracic Surgery, U. S. Navy and U. S. Veterans Administration.

5:00 P.M.

Discussers

RUDOLPH NISSEN, M.D., New York, N. Y.
EDWIN BOROS, M.D., New York, N. Y.
JOSEPH P. LYNCH, M.D., Brookline, Mass.

General Discussion

7:00 P.M.

ANNUAL BANQUET—HOTEL SOMERSET, Boston, Mass.

Presentation of 1949 Prize Award to JULIAN A. STERLING, M.D., Philadelphia, Pa., by DR. WILLIAM REID MORRISON, President, National Gastroenterological Association.

Invocation—RABBI HERMAN H. RUBENOWITZ, President, Rabbinical Association of Boston.

Speakers

MOST REV. RICHARD J. CUSHING, D.D., LL.D., Archbishop of Boston.

LORD ALFRED WEBB-JOHNSON, F.R.C.S., President, Royal College of Surgeons, London, England.

JAMES H. RAND, JR., President and Chairman of the Board of Remington Rand, Inc.

Benediction—RT. REV. RAYMOND A. HERON, D.D., Suffragan Bishop, Episcopal Diocese of Massachusetts.

FIFTH SESSION

WEDNESDAY MORNING, 26 OCTOBER 1949.

EARL J. HALLIGAN, M.D., President, New Jersey Chapter, National Gastroenterological Association, Chairman.

JOHN A. FOLEY, M.D., Dorchester, Mass., Co-chairman.

9:00 A.M.

13. "The Antibiotics in Gastrointestinal Diseases".

Speaker

MAXWELL FINLAND, M.D., Boston, Mass. Associate Professor of Medicine, Harvard Medical School; Chief, Fourth Medical Service, Boston City Hospital; Associate Physician, Thorndike Memorial Laboratory, Boston City Hospital and E. BUIST WELLS, M.D., Boston, Mass., Harvard Medical School and Boston City Hospital.

9:30 A.M.

Discussers

FRANCIS T. JANTZEN, M.D., Boston, Mass.
ALLAN NOVACK, M.D., Boston, Mass.

General Discussion

10:00 A.M.

14. "Some Pertinent Factors in Cholelithiasis".

Speaker

JOHN M. T. FINNEY, JR., B.S., M.D., Baltimore, Md., Assistant Professor of Surgery, Johns Hopkins Medical School; Visiting Surgeon, Union Memorial Hospital, Hospital for the Women of Maryland, and Children's Hospital School.

10:30 A.M.

Discussers

JOHN M. McGOWAN, M.D., Boston, Mass.
HOWARD A. BOUVE, M.D., Boston, Mass.
JOSEPH H. BURNETT, M.D., Boston, Mass.

General Discussion

11:00 A.M. Recess to visit the Commercial and Technical Exhibits.

11:20 A.M.

15. "Intestinal Obstruction".

Speaker

OWEN H. WANGENSTEEN, B.A., M.D., Ph.D., Minneapolis, Minn., Professor and Chairman, Department of Surgery, University of Minnesota School of Medicine.

11:50 A.M.

Discussers

JACOB FINE, M.D., Boston, Mass.
HERBERT G. DUNPHY, M.D., Brookline, Mass.
STANLEY NOWAK, M.D., Boston, Mass.

General Discussion

12:30 P.M.

LUNCHEON AND ROUND-TABLE DISCUSSION

Subject

"Preventive Medicine"

Speaker

HORACE W. SOPER, M.D., St. Louis, Mo., President-elect, National Gastroenterological Association.

VISIT THE EXHIBITS

SIXTH SESSION

WEDNESDAY AFTERNOON, 26 OCTOBER 1949.

1:30 P.M.

ANNUAL MEETING OF THE NATIONAL EXECUTIVE BOARD.

HORACE W. SOPER, M.D., President, National Gastroenterological Association, Chairman.

WILLIAM W. LERMANN, Vice-President, National Gastroenterological Association, Co-chairman.

2:00 P.M.

16. "Surgical Treatment of Portal Hypertension".

Speaker

C. STUART WELCH, M.S., Ph. D., M.D., Boston, Mass., Professor of Surgery, Tufts College Medical School; Surgeon-in-Chief, Pratt Diagnostic Clinic and New England Center Hospital; Surgeon, Carney Hospital; Surgeon-in-Chief of Surgery and Tumor Clinics, Boston Dispensary.

2:30 P.M.

Discussers

ROBERT R. LINTON, M.D., Brookline, Mass.

GEORGE MILLER, M.D., Boston, Mass.

General Discussion

3:00 P.M.

17. "Diabetes Mellitus".

Speaker

ELLIOTT P. JOSLIN, A.M., M.D., Sc.D., Boston, Mass., Clinical Professor of Medicine Emeritus, Harvard Medical School; Medical Director, George F. Baker Clinic, New England Deaconess Hospital; Consulting Physician, Boston City Hospital.

3:30 P.M.

Discussers

HENRY BAKER, M.D., Boston, Mass., Professor of Clinical Medicine, Tufts College Medical School.

J. EDWARD FLYNN, M.D., Boston, Mass.

DAVID ADLERSBERG, M.D., New York, N. Y.

General Discussion

4:00 P.M.

18. "The Effect of Resection of the Sympathetic and Parasympathetic Innervation of the Stomach Upon Gastric Acidity".

Speaker

REGINALD H. SMITHWICK, B.S., M.D., Boston, Mass., Professor of Surgery, Boston University School of Medicine; Surgeon-in-Chief, Massachusetts Memorial Hospital.

VISIT THE EXHIBITS

4:30 P.M.

Discussers

JOHN J. BYRNE, M.D., Boston, Mass.

General Discussion

The following papers will be read if time permits.

19. **"The Present Status of Flexible Tube Esophagoscopy".**
EDWIN BOROS, M.D., New York, N. Y.

20. **"Treatment of Peptic Ulcer with Vitamin D_2 and Extract of Gastroduodenal Mucosa".**
JUAN NASIO, M.D., Rosario, Argentina

21. **"The Role of the Liver and Lipoid Metabolism in the Production of Arteriosclerosis".**
LESTER M. MORRISON, M.D., Los Angeles, Calif.

22. **"Use of Water Soluble Chorophyll in Treatment of Long Standing Peptic Ulcers".**
WILLIAM OFFENKRANTZ, M.D., Brooklyn, N. Y.

LADIES COMMITTEE PROGRAM

MONDAY, 24 OCTOBER 1949

8:30 A.M. to 4:00 P.M.
Registration—Families of members and guests may register at the registration desk on the Convention floor.

6:00 P.M.
Convocation Ceremony.

6:30 P.M.
President's Annual Reception (*Admission by Card Only*).

TUESDAY, 25 OCTOBER 1949

8:30 A.M. to 4:00 P.M.
Registration—Families of members and guests may register at the registration desk on the Convention floor.

10:00 A.M.
Sightseeing—In private cars. Ladies and their guests are requested to make advance arrangements at the registration desk.

12:30 P.M.
Luncheon in honor of LADY WEBB-JOHNSON, at the Harvard Club. Reservations for the luncheon must be made in advance. No charge for wives of members. Guests $3.00 per person.

7:00 P.M.
Annual Banquet at the Somerset. Each lady attending will be presented with an orchid and will receive a valuable gift. Reservations must be made in advance.

VISIT THE EXHIBITS

TECHNICAL EXHIBITORS

(Those attending the Convention sessions are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with many new products and new equipment on display)

AMERICAN CYSTOSCOPE MAKERS, INC., New York City (Booth 7), extends a cordial invitation to members and guests to visit their booth. Their new operating gastroscope and examining gastroscopes of small diameter as well as other instruments of interest to the gastroenterologist will be on display.

AMES COMPANY, INC., Elkhart, Indiana (Booth 9). Their representatives will be glad to discuss Decholin tablets and Decholin Sodium ampuls, the standard hydrocholeretic agents for the treatment of billiary tract diseases. They will be demonstrating Clinitest and Hematest—simplified tests for the detection of urine-sugar and occult blood.

CHESTER A. BAKER LABORATORIES, INC., Boston, Mass. (Booth 6), New England's largest chain of professional pharmacies, manufacture and distribute several products of merit. You are invited to inspect these pharmaceutical and vitamin specialties. Chester A. Baker products are sold only through professional channels and are available to all druggists through their wholesaler.

BILHUBER-KNOLL CORP., Orange, N. J. (Booth 2). The fine medicinal chemicals which fill a most important place in the physician's armamentarium of dependable and useful medication, Bromural, Dilaudid, Metrazol, Octin, Tannalbin, Theocalcin, etc., are found at their booth. Visit their exhibit for the latest developments among these and their other prescription chemicals.

BRISTOL-MYERS COMPANY, New York City (Booth 8), cordially invites you to visit its booth where BRISTOL-MYERS' representatives will be in attendance to extend a hearty welcome to all visitors and to answer any questions pertaining to BUFFERIN, SAL HEPATICA and other famous products on display.

BROWN AND CONNOLLY INC., Boston, Mass. (Booth 14), will have a complete display of medical books of all publishers of interest to members of the National Gastroenterological Association.

CAMERON SURGICAL SPECIALTY COMPANY, Chicago (Booth 1). A new instrument is being introduced this year. A flexible esophagoscope known as the "Boroscope" and designed by Dr. Edwin Boros of New York. It is Cameron built, of stainless steel, black lined. No rubber "finger" at the end to obscure vision. It is passed under direct vision. Little need be said about the Omniangle Gastroscope. It is generally accepted throughout the world as the finest. Now with the new coated lens system which increases the lens illumination approximately 33 1/3%, no other gastroscope even approaches the efficiency of a Cameron Omniangle Gastroscope.

CIBA PHARMACEUTICAL PRODUCTS, INC., Summit, N. J. (Booth 20), invite you to visit their exhibit for latest information on CARMETHOSE, a non-systemic mucin like colloid with a high acid combining quality. Also featured will be TRASENTINE, a powerful antispasmodic with ability to abolish various spastic conditions of the hollow abdominal viscera; and TRASENTINE-Phenobarbital, a combination of TRASENTINE with the well known sedative Phenobarbital. Representatives in attendance will be very glad to answer questions on these and other Ciba products.

THE COCA-COLA COMPANY, Atlanta, Ga. Ice-cold Coca-Cola served through the cooperation and courtesy of The Coca-Cola Bottling Company of Boston and The Coca-Cola Company.

EDER INSTRUMENT COMPANY, Chicago, Ill. (Booth 15), will show an improved line of Gastroscopic equipment featuring the Eder-Chamberlin Model 400 Gastroscope and the Eder-Hufford Flexible Optical Esophagoscope. Also several newly developed instruments will be shown at their booth by its designer.

THE HARROWER LABORATORY, INC., Glendale, Calif. (Booth 5). The Harrower technical exhibit has three main points of interest: 1. Gastroscopic illustrations of the coating action of Mucotin, the mucin antacid, on a gastric ulcer. 2. The action of Prulose, a new corrective type treatment for functional constipation, is schematically presented. 3. The advantages of Hematocrin, a nutritional hematinic, are illustrated by means of the Kreb cycle. Literature and samples will be available at the booth.

IVES-CAMERON COMPANY, INC., New York City (Booth 18). Trained personnel will be present to explain an entirely new type of therapy. MONITAN is the first palatable liquid solution of sorbitan monooleate polyoxyethylene derivative, a wetting agent which quickly and easily emulsifies and reduces the particle size of fats and fat soluble materials. Each teaspoonful (5 cc.) of MONITAN contains, in a flavored, aqueous sugar and glycerin base, 1.5 Gm. of S.M.P.D. MONITAN is an exceptionally efficient emulsifier of fats and permits their greater absorption and utilization.

MALLON, DIVISION OF DOHO CHEMICAL CORPORATION, New York City Booth 13), is featuring the new topical anesthesia, RECTALGAN, for relief of pain and itching in hemorrhoids and pruritus. This new therapy enjoys many advantages over the outmoded rectal suppositories and ointments. Their representatives will be happy to explain, in detail, the workings of this medication.

THE NATIONAL DRUG COMPANY, Philadelphia (Booth 19). RESINAT—completely non-toxic, anion exchange resin antacid and pepsin inhibitor, and PROTINAL POWDER—delicious, micro pulverized, whole protein carbohydrate will be the featured products. Samples and literature will be available. Trained representatives will be on hand to answer all inquiries.

THE E. L. PATCH COMPANY, Stoneham, Mass. (Booth 12). Their representatives will be on hand to greet you and to answer your questions concerning Patch products. In addition to their old friend KONDREMUL, you will find an interesting demonstration of the new antacid, ALZINOX (brand of Dihydroxy Aluminum Aminoacetate), GLYTHEONATE (brand of Theophylline-Sodium Glycinate and SESTRAMIN, the natural estrogen product with vitamins. You are cordially invited to drop in and learn more about these service-rendering products.

RYSTAN COMPANY, INC., Mount Vernon, N. Y. (Booth 10). CHLORESIUM preparations contain the highly purified, therapeutically active water-soluble derivatives of Chlorophyll "a" ($C_{55}H_{72}O_5N_4Mg$). They are natural, nontoxic, biogenic healing and deodorizing agents for the treatment of lesions and inflammatory conditions of the gastrointestinal tract. They stimulate tissue to normal function, cause increase in the rate of healing and deodorize malodorous conditions by inhibiting the action of proteolytic bacteria.

SANDOZ PHARMACEUTICALS, New York City (Booth 11), invite all physicians to visit their booth where they are featuring Cafergone Tablets (E. C. 110), the first really effective oral Migraine treatment. When Histamine Cephalagia and tension headaches are impending or under way—Cafergone tablets are also indicated. Cafergone tablets either check the attack abruptly in the early stage, *actually preventing the onset of pain* or they interrupt the progress of the attack, *markedly shortening it and reducing it in severity*. Cafergone is available in tablet form only. Among other products displayed are: Dihydroergotamine (D.H.E.-45), Belladenal, Bellergal and Mesantoin.

SARATOGA SPRINGS AUTHORITY, Saratoga Springs, N. Y. (Booth 4). This exhibit consists of information explaining facilities available to the public at the New York State owned Saratoga Spa as a part of the State's public health service. The center theme is an aerial view of the 1200 acre reservation. It emphasizes treatments with naturally carbonated mineral waters, bottled waters, and recreational features. Saratoga Spa literature will be distributed by an attendant.

G. D. SEARLE & CO., Chicago (Booth 3), cordially invites you to visit their booth where their representatives will be happy to answer any questions regarding Searle Products of Research. Featured will be Dramamine for the prevention and active treatment of motion sickness; Ruphyllin, for abnormal capillary fragility; Hydryllin, new and effective antihistaminic, as well as such time-proven products as Searle Aminophyllin in all dosage forms, Metamucil, Ketochol, Floraquin, Kiophyllin, Diodoquin, Pavatrine and Pavatrine with Phenobarbital.

E. R. SQUIBB & SONS, New York City (Booth 23) will feature new professional Specialties. Also on display will be such widely accepted products as Crysticillin, Tolserol, Rubramin, Amnestrogen and the Penicillin Dispolator. The representatives in attendance will be pleased to discuss these and other Squibb products with you. Please visit their booth.

FREDERICK TROUT CO. INC., Mount Vernon, N. Y. (Booth 16).

U. S. VITAMIN CORPORATION, New York City (Booth 22). Exhibit demonstrates the greatest vitamin technicological advance of the present decade . . . "oil-in-water" multi-vitamin solutions . . . includes Vi-Syneral Injectable which makes available for the first time in pharmaceutical history, an *aqueous* parenteral multi-vitamin solution, ready for immediate injection; also, the *original oral* aqueous multi-vitamin formula, Vi-Syneral Vitamin Drops . . . since 1943. Professional samples and literature distributed on their full line of nutritional specialties, including Vi-Syneral Therapeutic, Tri-Sulfanyl, Methischol, Vi-Syneral capsules, Poly-B, Vi-Litron, Hypervitam, Lipo-Heplex and others.

WHITTIER LABORATORIES, Chicago (Booth 21). Visit their exhibit to discuss Resmicon, acid adsorbent demulcent. Resmicon is a new principle in peptic ulcer therapy, combining resin with gastric mucin in a single tablet. Thorough chewing of the tablet results in suspending the acid adsorptive and pepsin inactivating resin in the nuco-protective coating of mucin.

WINTHROP-STEARNS INC., New York City (Booth 17) extends a cordial invitation to visit its booth, where representatives will be on hand to serve you. Featured products will be Creamalin and Tricreamalate, nonabsorbable antacids; Demerol, powerful analgesic, spasmolytic and sedative; Parenamine, amino acids for intravenous use; Essenamine, tasteless protein concentrate for oral use; Pluraxin, high potency, therapeutic formula multiple vitamin capsules.

MOTION PICTURE PROGRAM

The following motion pictures will be shown daily at 9:30 A.M. and 2:30 P.M. in the special motion picture room.

1. **"The Role of Gastroscopy in the Diagnosis and Treatment of Gastric Pathology".**

LEO L. HARDT, M.D., Clinical Professor of Medicine, Loyola University School of Medicine, Chicago, Ill.

2. **"Ambulatory Gastroenterology".**

ALFRED J. CANTOR, M.D., Secretary, International Academy of Proctology, Flushing, N. Y.

3. **"Malnutrition in the Hospital Patient".**

EUGENE F. DUBOIS, M.D., Professor of Physiology, Cornell University Medical College, New York, N. Y.

ROBERT ELMAN, M.D., Professor of Clinical Surgery, Washington University School of Medicine, St. Louis, Mo.

HERBERT POLLACK, M.D., Associate Physician for Metabolic Diseases, Mt. Sinai Hosp., New York, N. Y.

POSTGRADUATE COURSE IN GASTROINTESTINAL SURGERY

SPONSORED BY THE

NATIONAL GASTROENTEROLOGICAL ASSOCIATION
POSTGRADUATE DIVISION TUFTS MEDICAL COLLEGE

AND THE

FIRST AND SECOND SURGICAL SERVICES BOSTON CITY HOSPITAL

27, 28, 29 OCTOBER 1949

OWEN H. WANGENSTEEN, B.A., M.D., Ph.D., Minneapolis, Minn., Professor and Chairman, Department of Surgery, University of Minnesota Medical School, Director.

New Cheever Amphitheater
Dowling Building
Boston City Hospital
Boston, Mass.

The last ten minutes of each presentation will be devoted to discussion and a question and answer period.

FIRST SESSION

THURSDAY MORNING, 27 OCTOBER 1949.

9:00 A.M.

1. **"Intestinal Obstruction."**

Speaker

OWEN H. WANGENSTEEN, B.A., M.D., Ph.D., Minneapolis, Minn., Professor, and Chairman, Department of Surgery, University of Minnesota Medical School.

10:00 A.M.

2. "Transpleural Bilateral Vagotomy with Resection of the Tenth Nerves."

Speaker

WILLIAM REID MORRISON, A.B., M.D., F.A.C.S., Boston, Mass., Surgeon-in-Chief, Second Surgical Service, Boston City Hospital and STANLEY MIKAL, M.D., A.B., Boston, Mass., Teaching Resident Surgeon, First Surgical Service, Boston City Hospital.

10:30 A.M.

3. "Traumatic Injuries of the Liver and Spleen."

Speaker

GEORGE W. PAPEN, M.D., F.A.C.S., Boston, Mass., Surgeon-in-Chief, First Surgical Service, Boston City Hospital.

11:00 A.M.

4. "Some Surgical Adventures."

Speaker

THE RT. HON. THE LORD ALFRED WEBB-JOHNSON, London, England, M.B.; Ch.B.; F.R.C.S.; Hon. LL.D.; Hon. F.A.C.S.; Hon. F.R.C.S.I.; Hon. F.R.C.S.E.; Hon. F.R.A.C.S.; Hon. F.F.R.P.S.; Knight Commander of the Victorian Order; Commander of the British Empire; Distinguished Service Order; Territorial Decoration; Deputy Lieutenant of the County of London; Doctor of Laws; President of the Royal College of Surgeons of London, England.

SECOND SESSION

THURSDAY AFTERNOON, 27 OCTOBER 1949.

1:00 P.M.

5. "The Diagnosis and Management of Splenomegaly."

Speaker

WILLIAM B. CASTLE, M.D., Boston, Mass., Director, Thorndike Laboratory and Director, Second and Fourth Medical Services, Boston City Hospital.

2:00 P.M.

6. "The Management of Stomach and Duodenal Diseases."

Speaker

FRANK H. LAHEY, M.D., Boston, Mass., Surgeon-in-Chief, New England Baptist Hospital; Surgeon, New England Deaconess Hospital; Director, Lahey Clinic.

3:00 P.M.

7. "Surgical Diseases of the Pancreas."

Speaker

ALEXANDER J. A. CAMPBELL, A.B., M.D., Boston, Mass., Assistant Visiting Surgeon, Boston City Hospital; Visiting Surgeon, New England Baptist Hospital, Brooks Hospital, Brookline; Mt. Auburn Hospital, Cambridge; Surgeon-in-Chief, Boston State Hospital; Consultant, Emerson Hospital, Concord; Assistant Professor of Surgery, Tufts College Medical School.

3:30 P.M.

8. "Surgical Repair of Femoral Hernia."

Speaker

NEWTON C. BROWDER, A.B., M.D., F.A.C.S., Boston, Mass., Visiting Surgeon, Boston City Hospital; Instructor in Surgery, Tufts Medical

School; IRVING MADOFF, A.B., M.D., Resident in Surgery, Boston City Hospital and FRANCIS A. KIRBY, A.B., M.D., Resident in Surgery, Boston City Hospital.

4:00 P.M.

9. "Observations on 3,000 Gastroscopies."

Speaker

CHARLES W. MCCLURE, A.B., M.D., F.A.C.P., Boston, Mass., Consulting Gastroscopist, Boston City Hospital; Consulting Gastroenterologist, Fifth and Sixth Medical Services, Boston City Hospital; Consulting Physician, Brooks Hospital, Brookline; I. R. JANKELSON, M.D., Boston, Mass., Assistant Professor of Medicine, Tufts College Medical School; Junior Visiting Physician and Gastroenterologist to Out-Patient Department, Boston City Hospital; Gastroscopist, Boston City Hospital; Visiting Physician, Jewish Memorial Hospital, Roxbury.

4:30 P.M.

10. "Ulcerative Colitis."

Speaker

DAVID D. BERLIN, M.D., Boston, Mass., Clinical Professor of Surgery, Tufts College Medical School; Visiting Surgeon, Beth Israel Hospital, Boston and Boston City Hospital.

THIRD SESSION
FRIDAY MORNING, 28 OCTOBER 1949.

9:00 A.M.

11. "The Antibiotics in Gastrointestinal Disease."

Speaker

MAXWELL FINLAND, M.D., Boston, Mass., Associate Professor of Medicine, Harvard Medical School; Chief, Fourth Medical Service, Boston City Hospital; Associate Physician, Thorndike Memorial Laboratory, Boston City Hospital.

10:00 A.M.

12. "Surgical Diseases of the Large Bowel."

Speaker

ALLAN L. DAVIS, M.D., F.A.C.S., Boston, Mass., Assistant Professor of Surgery, Tufts Medical School; Visiting Surgeon, Boston City Hospital.

10:30 A.M.

13. "The Abdominal Incision."

Speaker

D. JOSEPH DUGGAN, A.B., M.D., F.A.C.S., Boston, Mass., Senior Surgeon, Boston City Hospital.

11:00 A.M.

14. "Lesions of the Small Bowel."

Speaker

CHARLES A. LAMB, A.B., M.D., F.A.C.S., Boston, Mass., Boston City Hospital, New England Deaconess Hospital and Newton-Wellesley Hospital.

11:30 A.M.

15. "An Appraisal of Current Methods for the Treatment of Portal Hypertension."

Speaker

C. STUART WELCH, M.S., M.D., Ph.D., Boston, Mass., Professor of Surgery and Head of Department, Tufts Medical School; Surgeon-in-Chief, New England Center Hospital, Boston, Mass.

FOURTH SESSION
FRIDAY AFTERNOON, 28 OCTOBER 1949.

1:00 P.M.

16. "Diabetes Mellitus and Gastrointestinal Surgery."

Speaker

ELLIOTT P. JOSLIN, A.M., M.D., Sc.D., Boston, Mass., Clinical Professor of Medicine, Emeritus, Harvard Medical School; Medical Director, George F. Baker Clinic, New England Deaconess Hospital; Consulting Physician, Boston City Hospital.

2:00 P.M.

17. "Surgical Aspects of Diverticulitis of the Sigmoid."

Speaker

J. EDWARD FLYNN, A.B., M.D., F.A.C.S., Boston, Mass., Instructor in Surgery, Tufts Medical School; Assistant Visiting Surgeon, Boston City Hospital; Assistant Visiting Surgeon, St. Elizabeth's Hospital; Attending Surgeon, Veterans Administration Hospital, West Roxbury; Consultant Surgeon, Boston State Hospital.

2:30 P.M.

18. "Acute Perforated Peptic Ulcer."

Speaker

CHARLES G. SHEDD, M.D., F.A.C.S., Boston, Mass., Assistant Visiting Surgeon, Second Surgical Service, Boston City Hospital and I. SCHWARTZ, M.D., Boston, Mass., Resident Surgeon, First Surgical Service (Tufts Medical School Teaching Service), Boston City Hospital.

3:00 P.M.

19. "Gastrojejunocolic Fistula."

Speaker

JOSEPH TARTAKOFF, M.D., F.A.C.S., Boston, Mass., Assistant Professor of Surgery, Tufts Medical School; Junior Visiting Surgeon, Boston City Hospital.

3:30 P.M.

20. "Congenital Pyloric Stenosis."

Speaker

JOHN HOWLAND CRANDON, A.B., M.D., Boston, Mass., Instructor in Surgery, Tufts Medical School; Assistant to Visiting Surgeon, Boston City Hospital; Junior Surgeon, Mt. Auburn Hospital, Cambridge.

4:00 P.M.

21. "Recurrent Inguinal Hernia."

Speaker

ROGER T. DOYLE, M.D., F.A.C.S., Boston, Mass., Junior Visiting Surgeon, Boston City Hospital; Assistant Visiting Surgeon, St. Elizabeth's Hospital.

4:30 P.M.

22. "Complications of Acute Appendicitis."

Speaker

HARRISON E. KENNARD, A.B., M.D., Boston, Mass., Assistant in Surgery, Tufts College Medical School; Surgical Staff, Boston City Hospital.

FIFTH SESSION
SATURDAY MORNING, 29 OCTOBER 1949.

9:00 A.M.

23. "Gallbladder Disease."

Speaker

THOMAS W. WICKHAM, A.B., M.D., M.A., D.Sc., Boston, Mass., Surgeon-in-Chief, Fourth Surgical Service, Boston City Hospital.

10:00 A.M.

24. "X-rays of Gastrointestinal Tract."

Speaker

MAX RITVO, A.B., M.D., Boston, Mass., Assistant Professor, Harvard Medical School; Instructor, Tufts Medical School; Director, Department of Radiology, Boston City Hospital.

11:00 A.M.

25. "Relationship of Vascular Disease to the Gastrointestinal Tract."

Speaker

EUGENE E. O'NEIL, A.B., M.D., Boston, Mass., Professor of Clinical Surgery, Boston University, School of Medicine; Visiting Surgeon, Boston City Hospital, Faulkner Hospital; St. Elizabeth's Hospital.

11:30 A.M.

26. "Upper-Gastrointestinal Hemorrhage."

Speaker

FRANKLIN W. WHITE, S.B., M.D., Boston, Mass., Consulting Physician, Boston City Hospital; THOMAS C. CHALMERS, A.B., M.D., Boston, Mass., Instructor of Medicine, Harvard University; Junior Visiting Physician, Boston City Hospital, Mt. Auburn Hospital, Cambridge and NORMAN ZAMCHECK, A.B., M.D., Boston, Mass., Research Fellow, Thorndike Memorial Laboratory and Department of Medicine, Harvard Medical School; Assistant in Medicine, Boston City Hospital.

SIXTH SESSION
SATURDAY AFTERNOON, 29 OCTOBER 1949.

1:00 P.M.

27. "Surgical Lesions of the Esophagus."

Speaker

JOHN W. STRIEDER, M. D., Boston, Mass. Associate Professor of Thoracic Surgery, Boston University School of Medicine; Surgeon-in-Chief for Thoracic Surgery, Boston City Hospital Sanatorium Division, Massachusetts Memorial Hospitals, Newton-Wellesley Hospital.

2:00 P.M.

28. "Anesthesia in Gastrointestinal Surgery."

Speaker

SIDNEY CUSHING WIGGIN, B.S., M.D., Boston, Mass., Chief Anesthetist, Boston City Hospital; Consulting Anesthetist, Bedford Veterans Hospital and Brighton Marine Hospital and PETER SAUNDERS, M.D., Boston, Mass., Assistant in Anethesiology, Tufts College Medical School and Boston University Medical School; Junior Visiting Anesthetist, Boston City Hospital.

3:00 P.M.

29. "Carcinoma of the Mouth."

Speaker

CHARLES C. LUND, A.B., M.D., Boston, Mass., Assistant Professor of Surgery, Harvard Medical School; Visiting Surgeon, Boston City Hospital.

3:30 P.M.

30. "The Nutritional Care of the Surgical Patient."

Speaker

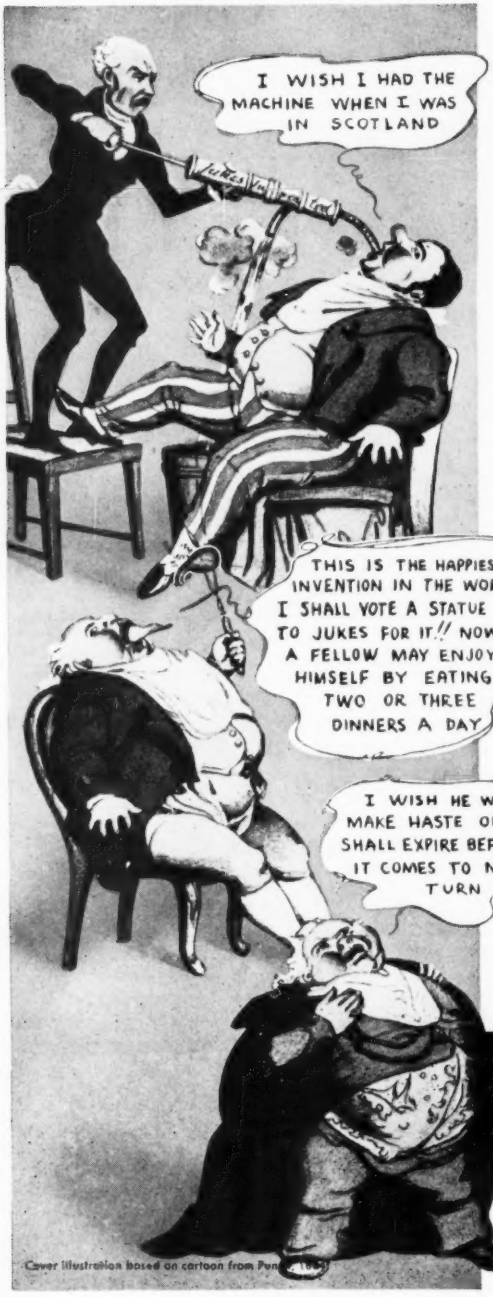
CHARLES S. DAVIDSON, M.D., Boston, Mass., Associate in Medicine, Harvard Medical School; Associate Physician, Thorndike Memorial Hospital, Boston City Hospital; Associate Director, Second and Fourth Medical Services, Boston City Hospital and RICHARD D. ECKHARDT, M.D., Boston, Mass., Associate, Department of Internal Medicine, State University of Iowa, Iowa City, Iowa.

4:00 P.M.

31. "Lesions of the Common Bile Duct."

Speaker

JOHN M. McGOWAN, B.S., M.D., C.M., M.S., F.A.C.S., F.R.C.S., Boston, Mass., Instructor in Surgery, Tufts Medical School; Surgeon, Boston City Hospital and Quincy City Hospital.



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SEE OUR EXHIBIT AT BOOTH XIV

NEWS NOTES

Fourteenth Annual Convention

The Program for our Fourteenth Annual Convention is bound into this issue of The Review of Gastroenterology and will not be mailed separately to the members of the Association. Additional copies are available from the Headquarters Office, 1819 Broadway, New York 23, N. Y. The Program will also be available at the registration desk on the Convention floor.

The Program Committee consisting of Drs. William Reid Morrison, Charles W. McClure, I. R. Jankelson, Anthony Bassler, Roy Upham and Samuel Weiss is to be congratulated on the excellence of the program to be presented.

1949 Prize Award to Dr. Julian A. Sterling

The National Gastroenterological Association takes pleasure in announcing that its 1949 Prize Award and a Certificate of Merit is being presented to Dr. Julian A. Sterling of Philadelphia, Pa. for his paper on "The Termination of the Common Bile Duct".

Certificates of Merit were also awarded by the judges of the contest to Dr. R. A. Jamieson of Glascow, Scotland for his paper on "Observations on an Isolated Gastric Pouch in Man"; Dr. René Schubert of Tubingen, Germany for his paper "A Novel Means of Detoxicating the Body by Replacing the Bile-Liver System by the Kidneys as Organs of Excretion of Substances Bound to Artificial Colloids"; Dr. Mario Stefanini of Milwaukee, Wisconsin for his paper on "Hyperbilirubinemic Effect of Sodium Nicotinate" and Dr. A. L. Soresi of New York, N. Y. for his paper on "Depressionless Relaxation for Gastrointestinal Surgery".

Dr. Sterling's paper will appear in its entirety in a forthcoming issue of The Review of Gastroenterology.

Registration

All those attending the Convention are requested to register and receive their identification badges. Ladies are also invited to register. No one will be admitted to the exhibits or the sessions without a badge.

Annual Meeting of the National Gastroenterological Association

The Annual Meeting of the National Gastroenterological Association will be held at the Hotel Somerset in Boston, Mass., at 5:30 p.m. on Monday, 24 October 1949, the first day of the Fourteenth Annual Convention.

Members of the Association are invited to attend and participate in the business meeting.

The proposed revision of the constitution will be acted upon at that time.

Convocation Ceremony

The Convocation Ceremony, at which time Fellowship certificates will be presented in person to those newly elected Fellows and those who have been advanced to Fellowship during the past year, will follow the Annual Meeting of the Association at 6:00 p.m. on Monday, 24 October 1949.

Annual Meeting of the National Council

The National Council of the National Gastroenterological Association will hold its Annual Meeting immediately preceding the Fourth Session of the Convention on Tuesday afternoon, 25 October 1949 at 1:00 p.m.

Officers of the Association, Presidents of the Chapters and delegates are requested to attend.

National Executive Board Meeting

The Annual Meeting of the National Executive Board will be held immediately following the fifth session of the Convention on Wednesday afternoon, 26 October 1949 at 1:30 p.m.

President's Annual Reception

The President's Annual Reception will be held immediately following the Convocation Ceremony on Monday evening, 24 October 1949 at 6:30 p.m. Members of the Association and their friends, as well as those present with the technical exhibits are cordially invited to attend. Admission cards may be secured at the time of registration at the registration desk.

This year we are again fortunate in having this annual event sponsored by Winthrop-Stearns, Inc., who have so graciously cooperated with us in making this affair an annual social highlight.

Annual Banquet

The Annual Banquet of the National Gastroenterological Association will be held at the Hotel Somerset in Boston, Mass. on Tuesday evening, 25 October 1949 at 7:00 p.m. to be preceded by cocktails.

The cost of the dinner will be $15.00 per couple and reservations are available through the National Gastroenterological Association, 1819 Broadway, New York 23, N. Y. or at the registration desk.

A valuable gift will be given to each person attending the Banquet through the courtesy of Mr. James H. Rand, Jr., President and Chairman of the Board of Remington Rand, Inc. Each lady attending the Banquet will receive, in addition, an orchid in an attractive lapel pin vase.

The 1949 Prize Award for the best unpublished contribution on Gastroenterology or an allied subject will be awarded to Dr. Julian A. Sterling of Philadelphia,

Pa. for his paper "The Termination of the Common Bile Duct". The first prize, consisting of a check for $100.00 and a Certificate of Merit, will be awarded to Dr. Sterling by Dr. William R. Morrison, President of the National Gastroenterological Association.

Among the others to speak at the banquet will be Lord Alfred-Webb Johnson, President of the Royal College of Surgeons in England, Mr. James H. Rand, Jr., Chairman and President of the Board of Remington Rand, Inc. and the Most Rev. Richard J. Cushing, Archbishop of Boston.

The invocation will be by Rabbi Herman H. Rubenowitz, President of the Rabbinical Association of Boston and the benediction will be delivered by the Rt. Rev. Raymond A. Heron, Suffragan Bishop of the Episcopal Diocese of Massachusetts.

A medallion will be presented to Lord Alfred Webb-Johnson on behalf of the National Gastroenterological Association.

Course in Gastrointestinal Surgery

Immediately following our 14th Annual Convention, the National Gastroenterological Association, in conjunction with the Postgraduate Division of the Tufts Medical College and the First and Second Surgical Services of the Boston City Hospital, is conducting a three day course in Gastrointestinal Surgery.

This course will be under the personal direction of Dr. Owen H. Wangensteen, Professor and Chairman of the Department of Surgery of the University of Minnesota Medical School. Participating in the course will be Lord Alfred Webb-Johnson, President of the Royal College of Surgeons of London, England as well as other distinguished guest lecturers. Dr. William Reid Morrison is chairman of the arrangements committee.

Enrollment for the course has closed and admission is limited to those holding matriculation cards indicating that they have paid the $35.00 fee and have been accepted.

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relieves pain The anion-exchange polyamine resin in Resmicon rapidly inactivates HCl and pepsin by physical adsorption. Pain is swiftly relieved.

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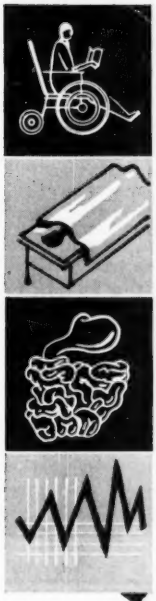

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supply man-sized vitamin potencies for the big jobs—severe vitamin deficiencies; convalescence from serious illness or major surgery; gastrointestinal dysfunctions impairing absorption or utilization of dietary factors; severe infections.

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Vitamin B_1	25 mg.
Vitamin B_2	15 mg.
Nicotinamide	100 mg.
Folic Acid (Vitamin B_c)	2.5 mg.
Vitamin B_6	1 mg.
Pantothenic Acid (as sodium salt)	10 mg.
Vitamin C	150 mg.

Dose: 1 or 2 KAPSEALS daily for 10 to 14 days.
Packaging: Bottles of 100 and 1000 KAPSEALS.

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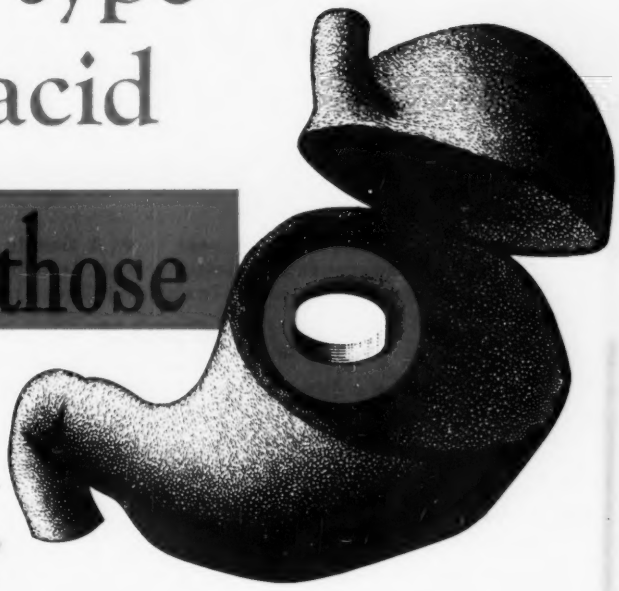

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Carmethose promptly lowers gastric acidity, and its protective tenacious coating has been observed in the stomach for as long as three hours.[1]

Adult dose is 2 to 4 tablets or teaspoonfuls 4 times daily between meals.

Carmethose Tablets: *sodium carboxymethylcellulose, 225mg. and magnesium oxide, 75mg. Bottles of 100*

Carmethose Liquid: *5% concentration of sodium carboxymethylcellulose. Bottles of 12 oz.*

Advantages over adsorbent gels:

1. *Non-constipating*—hydrophilic gel promotes normal elimination.[1, 2]
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3. *Palatable*—small, easily swallowed tablets and pleasantly flavored liquid—preferred by patients.[2]

Advantages over soluble alkalis:

1. *No acid rebound*—effectively inhibits acid-pepsin activity, with no secondary hypersecretion.
2. *Protective coating*—mucin-like gel is rapidly formed and clings to ulcer crater and gastric mucosa.
3. *Non-systemic*—cannot disturb acid-base balance because it is nonabsorbable.

1. Brick, I.B.: Amer. J. Dig. Dis., *In Press* 2. Bralow, Spellberg & Necheles: Scientific Exhibit #1112, A.M.A. Annual Session 1949

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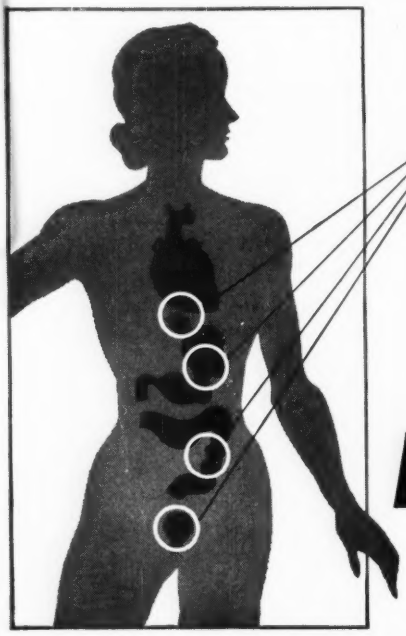

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New oral management revealed*

Peptic Ulcer

Duodenum desiccated and defatted by a special process (Viodenum), is being evaluated clinically in the treatment of ulcerative colitis and peptic ulcer, on the hypothesis that it may render the mucosa more resistant to chemical and mechanical injury and improve the mechanisms of repair... *Preliminary observations indicate a thorough clinical trial with Viodenum in those patients refractory to other treatment.* The medication should be viewed as supportive therapy to the usual treatment.

Viodenum

Provided in powder or 10 grain tablets

Raw* duodenum, desiccated and defatted at 37° C.

*"Rawness" measured by: presence of enzymes identified with the raw tissue of the freshly killed animal

Administration of Viodenum may result in—

relief of symptoms
diminishing bowel movements
diminishing exacerbations
normal stool consistency
gain in weight

*References:

*Streicher, M. H. F., Grossman, M. I., and Ivy, A. C., Gastroenterology, **12,** 371 (1949)

*Raimondi, P. J., Goetzl, F. R., Permanente Foundation Medical Bulletin, **7,** 1 (1949)

Gill, A. M., Lancet, **2,** 202 (1945); Proc. Roy. Soc. Med., **39,** 517 (1946)

Rivers, A. B., Am. J. Dig. Dis., **2,** 189 (1935)

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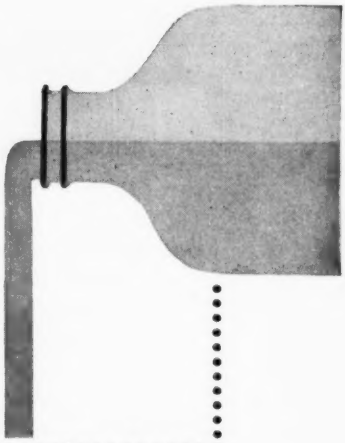

Solution Choline Gluconate-C.S.C. represents a significant advance in choline therapy. Its citrus orange flavor is unusually delightful, making it acceptable even over the prolonged periods required in the treatment of many hepatic disorders. This preparation, which replaces Syrup Choline Bicarbonate-C.S.C., contains 62.5% choline gluconate or the equivalent of 25% choline base, the highest choline content of any choline solution available today. Furthermore, its low cost to the patient in terms of choline content, makes it outstandingly economical.

Solution Choline Gluconate-C.S.C. is indicated in the treatment of all hepatic, precirrhotic, and cirrhotic conditions in which choline is of value. It is best taken diluted with a half glass of water.

Now available on prescription at all pharmacies in one pint bottles.

C.S.C. Pharmaceuticals

A DIVISION OF COMMERCIAL SOLVENTS CORPORATION, 17 E. 42ND ST., NEW YORK 17, N. Y.

Solution
CHOLINE GLUCONATE

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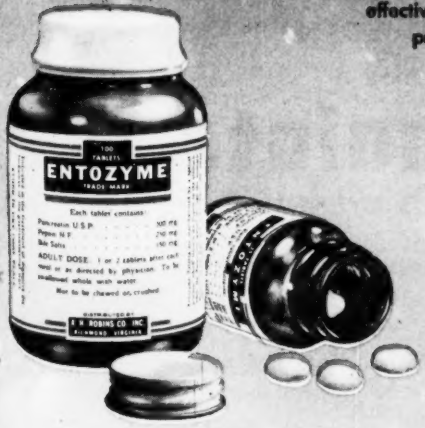
announcing another Robins achievement

NEW TRIPLE-ENZYME DIGESTANT

with unique Peptomatic* Action!

Gastrically soluble outer shell contains pepsin; enterically coated core contains pancreatin and bile salts.

By the development of an entirely new type of coated tablet, consisting of a gastrically soluble outer shell containing pepsin, and an enterically coated core containing pancreatin and bile salts—Robins (with their new product Entozyme) now makes it possible to release these three important digestants *in fully active form* to that part of the gastrointestinal tract where pH conditions for optimum activity prevail. Clinical research[1] indicates that Entozyme's greatest field of usefulness is in chronic cholecystitis, post-cholecystectomy syndrome, subtotal gastrectomy, infectious hepatitis, pancreatitis and chronic dyspepsia—where its unique selective therapy restores more nearly physiological conditions in the gastrointestinal tract. It is also highly effective in nausea, anorexia, belching, flatulence and pyrosis. In peptic ulcer patients, too, pancreatin-pepsin therapy has produced excellent results.[2]



FORMULA: Each specially constructed tablet contains Pancreatin, U.S.P., 300 mg.; Pepsin, N.F., 250 mg.; Bile Salts, 150 mg.

DOSAGE: 1 or 2 tablets after each meal, or as directed by physician, without crushing or chewing

SUPPLIED: Bottles of 25 and 100.

REFERENCES: 1. McGavack, T. H. and Klotz, S. D.: Bull. Flower Fifth Ave. Hosp., 9:61, 1946. 2. Weissberg, J., McGavack, T. H. and Boyd, Linn J.: Am. J. Digest. Dis., 15:332, 1948.

**A coined word to describe the unique mechanical action of Entozyme Tablet—whereby pepsin is released only in the stomach, and pancreatin and bile salts only in the small intestine.*

A. H. ROBINS COMPANY, INC. • RICHMOND 20, VIRGINIA

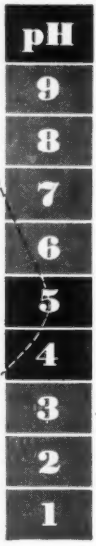
Ethical Pharmaceuticals of Merit since 1878

ENTOZYME

"safety zone"

therapy of

PEPTIC ULCER



The gastric pH range which is safe for the peptic ulcer patient lies between 4 and 5. In this "safety zone" there is neither pepsin activity (which may cause continued erosion or bleeding) nor stimulation of excess acid production.

Tricreamalate, a balanced blend of aluminum hydroxide gel with magnesium trisilicate reduces acidity within the stomach to pH 4 to 5. Absolute neutrality is not reached. Hence, there is no stimulus to "acid rebound" and no alkalosis.

Through the formation of a protective coating and a mild astringent effect, nonabsorbable Tricreamalate is soothing to the irritated gastric mucosa, relieves gastric pain and heartburn, and aids in healing peptic ulceration as well as in preventing recurrence.

Tricreamalate

Aluminum Hydroxide Gel with Magnesium Trisilicate

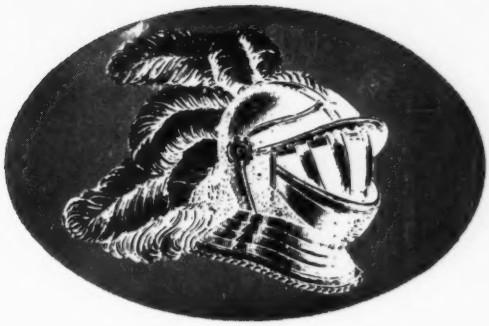
LIQUID
Bottles of 12 fl. oz.
TABLETS
Tins of 12
Bottles of 100 and 500

Dose: 1 or 2 teaspoonfuls or tablets every 2 to 4 hours.

Winthrop-Stearns INC.
NEW YORK 13, N. Y. WINDSOR, ONT.

Tricreamalate, trademark

SEE OUR EXHIBIT AT BOOTH 17



Protective covering . . .

Gelusil Antacid Adsorbent*, an especially prepared, nonreactive aluminum hydroxide gel, protects the inflamed or ulcerated areas of the gastric mucosa against injury by the acid gastric juice. Unlike most aluminum hydroxide preparations, *Gelusil* Antacid Adsorbent* is nonconstipating.[1]

Gelusil* 'Warner'

Antacid Adsorbent

Gelusil*
Antacid
Adsorbent

provides rapid and sustained relief of gastric hyperacidity and is particularly effective as an adjuvant in the medical management of gastric or duodenal ulcer.

is available as a pleasant-tasting liquid or tablet.

Liquid—6 and 12 fluidounces.

Tablets, individually wrapped in cellophane—boxes of 50 and 100 tablets. Also bottles of 1000.

[1]Rossien, A. X., and Victor, A. W.: The Influence of An Antacid on Evacuation of the Bowels and the Fecal Column, *Am. J. Dig. Dis.*, *14*:226, July, 1947.

William R. Warner & Co., Inc.

New York St. Louis

*T. M. Reg. U. S. Pat. Off.

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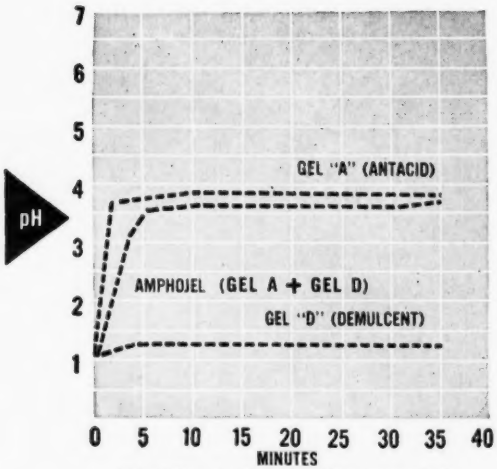

The reaction rate of Amphojel and its component gels.

the double action of AMPHOJEL®

antacid
demulcent

Amphojel – Aluminum Hydroxide Gel, Alumina Gel Wyeth – is unique because it is a colloidal mixture of two essentially different types of alumina gel, one having an antacid effect . . . the other a demulcent action.

The "antacid gel" instantly stops gastric corrosion and establishes a mildly acid environment.

The "demulcent gel" provides a prolonged local protective effect, and might be likened to a "mineral mucin."

Thus, through its double action, Amphojel gives you an excellent preparation for use in the management of peptic ulcer.

Wyeth®

WYETH INCORPORATED, PHILADELPHIA 3, PA.

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